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> d his

     (FILE 'REGISTRY' ENTERED AT 09:30:07 ON 10 JUN 2002)
                DEL HIS Y
                ACT GUPTA/A
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L2   (   1041853)SEA FILE=REGISTRY ABB=ON  PLU=ON  ^[AGPSTIMLVPWY]/SQSP
L3   (    198202)SEA FILE=REGISTRY ABB=ON  PLU=ON  L2 AND L1
L4   (    125102)SEA FILE=REGISTRY ABB=ON  PLU=ON  [AGPST]^/SQSP|[IMLVPWY]^/SQSP
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L6   (   2895101)SEA FILE=REGISTRY ABB=ON  PLU=ON  (CYCLIC OR CYCLO)
L7   (     14745)SEA FILE=REGISTRY ABB=ON  PLU=ON  CYCLIC/NTE
L8   (   2896366)SEA FILE=REGISTRY ABB=ON  PLU=ON  L6 OR L7
L9        115263 SEA FILE=REGISTRY ABB=ON  PLU=ON  L5 NOT L8
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     FILE 'HCAPLUS' ENTERED AT 09:34:06 ON 10 JUN 2002
L10        43218 S L9
L11          727 S CHARGE? (L) (AMINO ACID#)
L12         2300 S (CHARGE? (2A) (AMINO ACID#))/AB
L13           17 S L11  AND L10
L14           92 S L12 AND L10
L15         1713 S (CHARGE? (W) (AMINO ACID#))/AB
L16           73 S L15 AND L10
L17       102011 S PEPTIDE#/CW
L18           10 S L13 AND L17
L19            6 S L13 AND L16
L20           17 S L13 OR L19
L21         3413 S HYDROPHOBIC (L) AMINO ACID# OR (HYDROPHOBIC (2W) AMINO ACID#)
L22          125 S L21 AND  (L15 OR L11)
L23        96837 S T (L) (CELL# OR LYMPHOCYTE?)
L24            6 S L23 AND L22
L25       203849 S PEPTIDE#
L26        24723 S L25 AND L10
L27           70 S L25 AND (L13 OR L14)
L28           11 S L25 AND L13
L29           46 S L25 AND L16
L30       602878 S SEQUENCE# OR  SEQUENCE#/AB
L31           28 S L29 AND L30
L32           17 S L28 OR L20
L33           28 S L31 NOT L32

     FILE 'REGISTRY' ENTERED AT 09:46:03 ON 10 JUN 2002

     FILE 'HCAPLUS' ENTERED AT 09:46:12 ON 10 JUN 2002
L34           45 S L32 OR L33
L35           11 S L34 AND (TCR OR L23)
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=> fil reg
FILE 'REGISTRY' ENTERED AT 09:50:00 ON 10 JUN 2002
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STRUCTURE FILE UPDATES:     7 JUN 2002  HIGHEST RN 427375-75-5
DICTIONARY FILE UPDATES:    7 JUN 2002  HIGHEST RN 427375-75-5

TSCA INFORMATION NOW CURRENT THROUGH January 7, 2002

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

Calculated physical property data is now available.  See HELP PROPERTIES
for more information.  See STNote 27, Searching Properties in the CAS
Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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L8   (   2896366)SEA FILE=REGISTRY ABB=ON  PLU=ON  L6 OR L7
L9        115263 SEA FILE=REGISTRY ABB=ON  PLU=ON  L5 NOT L8
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→ sequence search

↳ they do not index charged a.acids in sequences. Need to look for them in C# file.

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FILE 'HCAPLUS' ENTERED AT 09:50:07 ON 10 JUN 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 10 Jun 2002  VOL 136 ISS 24
FILE LAST UPDATED: 7 Jun 2002  (20020607/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

CAS roles have been modified effective December 16, 2001.  Please
check your SDI profiles to see if they need to be revised.  For
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information on CAS roles, enter HELP ROLES at an arrow prompt or use
the CAS Roles thesaurus (/RL field) in this file.
'OBI' IS DEFAULT SEARCH FIELD FOR 'HCAPLUS' FILE

=> d his l10-

     (FILE 'REGISTRY' ENTERED AT 09:30:07 ON 10 JUN 2002)
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     FILE 'HCAPLUS' ENTERED AT 09:34:06 ON 10 JUN 2002
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L12         2300 S (CHARGE? (2A) (AMINO ACID#))/AB
L13           17 S L11  AND L10
L14           92 S L12 AND L10
L15         1713 S (CHARGE? (W) (AMINO ACID#))/AB
L16           73 S L15 AND L10
L17       102011 S PEPTIDE#/CW
L18           10 S L13 AND L17
L19            6 S L13 AND L16
L20           17 S L13 OR L19
L21         3413 S HYDROPHOBIC (L) AMINO ACID# OR (HYDROPHOBIC (2W) AMINO ACID#)
L22          125 S L21 AND  (L15 OR L11)
L23        96837 S T (L) (CELL# OR LYMPHOCYTE?)
L24            6 S L23 AND L22
L25       203849 S PEPTIDE#
L26        24723 S L25 AND L10
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L29           46 S L25 AND L16
L30       602878 S SEQUENCE# OR  SEQUENCE#/AB
L31           28 S L29 AND L30
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L33           28 S L31 NOT L32

     FILE 'REGISTRY' ENTERED AT 09:46:03 ON 10 JUN 2002

     FILE 'HCAPLUS' ENTERED AT 09:46:12 ON 10 JUN 2002
L34           45 S L32 OR L33
L35           11 S L34 AND (TCR OR L23)
                 SELECT RN L35 HIT 1-11

     FILE 'REGISTRY' ENTERED AT 09:50:00 ON 10 JUN 2002

     FILE 'HCAPLUS' ENTERED AT 09:50:07 ON 10 JUN 2002

=> d .ca l34 1-45

L34  ANSWER 1 OF 45  HCAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:872975  HCAPLUS
DOCUMENT NUMBER:          136:36354
TITLE:                    Preparation of cell surface antigen specific
                          monoclonal antibody fusion proteins
INVENTOR(S):              Shimizu, Nobuyoshi; Takayanagi, Atsushi
PATENT ASSIGNEE(S):       Keio University, Japan
SOURCE:                   Jpn. Kokai Tokkyo Koho, 24 pp.
                          CODEN: JKXXAF
DOCUMENT TYPE:            Patent
LANGUAGE:                 Japanese
FAMILY ACC. NUM. COUNT:   1
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too many sequences to printout

narrowed to references with TCR or Tcells

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2001333780	A2	20011204	JP 2000-158575	20000529

AB Disclosed is construction of a novel DNA transport system. The system comprises human-type single chain immunoporter monoclonal antibody-body protein fusion protein and fusion protein-DNA complex. The single chain monoclonal antibody fusion proteins are specific to cell surface antigen such as epidermal growth factor receptor, and are capable of binding to genetic element such as antitumor genes. The immunoporter monoclonal antibody fusion proteins are characterized by contg. less than 90 pos. (or neg.) **charged amino acids** to bind with genetic element that would be transported by endocytosis. The novel gene carrier is esp. useful for gene therapy of cancer.

IC ICM C12N015-09
ICS A61K038-00; A61K039-395; A61P035-00; C07K014-00; C07K016-28; C07K019-00; C12P021-02; C12P021-08; C12R001-19; C12R001-84

CC 15-3 (Immunochemistry)
Section cross-reference(s): 3

IT **Amino acids**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study) (neg. or pos. **charged**; cell surface antigen-specific monoclonal antibody fusion proteins for transporting DNA and gene therapy)

IT 21743-34-0 177746-73-5 191916-06-0 **206750-67-6** 364319-91-5
380151-67-7 380151-68-8 380151-69-9 380151-70-2 380151-71-3
380151-72-4 380151-73-5 380289-87-2 380289-88-3 380289-89-4
380289-90-7 380289-91-8 380289-92-9 380289-93-0 380289-94-1
380289-95-2 380289-96-3 380289-97-4
RL: PRP (Properties)
(unclaimed sequence; prepn. of cell surface antigen specific monoclonal antibody fusion proteins)

L34 ANSWER 2 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:831236 HCAPLUS
DOCUMENT NUMBER: 136:323610
TITLE: **Peptide** binding characteristics of the non-classical class Ib MHC molecule HLA-E assessed by a recombinant random **peptide** approach
AUTHOR(S): Stevens, James; Joly, Etienne; Trowsdale, John; Butcher, Geoffrey W.
CORPORATE SOURCE: Lab. of Functional Immunogenetics, The Babraham Institute, Cambridge, CB2 4AT, UK
SOURCE: BMC Immunology [online computer file] (2001), 2, No pp. given
CODEN: BIMMCV; ISSN: 1471-2172
URL: http://www.biomedcentral.com/1471-2172/2/5
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English

AB Background: Increasing evidence suggests that the effect of HLA-E on Natural Killer (NK) cell activity can be affected by the nature of the peptides bound to this non-classical, MHC class Ib mol. However, its reduced cell surface expression, and until recently, the lack of specific monoclonal antibodies hinder studying the peptide-binding specificity HLA-E. Results: An in vitro refolding system was used to assess binding of recombinant HLA-E to either specific peptides or a nonamer random peptide library. Peptides eluted from HLA-E mols. refolded around the nonamer library were then used to det. a binding motif for HLA-E.

Hydrophobic and non-**charged amino acids** were found to predominate along the peptide motif, with a leucine anchor at P9, but surprisingly there was no methionine preference at P2, as suggested by previous studies. Conclusions: Compared to the results obtained with rat classical class Ia MHC mols., RTI-AIc and RTI-Au, HLA-E appears to refold around a random peptide library to reduced but detectable levels, suggesting that this mol.'s specificity is tight but probably not as exquisite as has been previously suggested. This, and a previous report that it can assoc. with synthetic peptides carrying a viral **sequence**, suggests that HLA-E, similar to its mouse counterpart (Qa-Ib), could possibly bind peptides different from MHC class I leader peptides and present them to T lymphocytes.

CC 15-2 (Immunochemistry)

ST HLA E refolding **peptide** binding

IT Structure-activity relationship
(HLA-E-binding; **peptide** binding characteristics of non-classical class Ib MHC mol. HLA-E assessed by a recombinant random **peptide** approach)

IT Histocompatibility antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HLA-E; **peptide** binding characteristics of non-classical class Ib MHC mol. HLA-E assessed by a recombinant random **peptide** approach)

IT Lymphocyte
(natural killer cell; **peptide** binding characteristics of non-classical class Ib MHC mol. HLA-E assessed by a recombinant random **peptide** approach)

IT Protein folding
(**peptide** binding characteristics of non-classical class Ib MHC mol. HLA-E assessed by a recombinant random **peptide** approach)

IT **Peptides**, biological studies
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(**peptide** binding characteristics of non-classical class Ib MHC mol. HLA-E assessed by a recombinant random **peptide** approach)

IT **159471-80-4 202657-60-1** 310899-73-1
310900-06-2 415706-85-3
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(**peptide** binding characteristics of non-classical class Ib MHC mol. HLA-E assessed by a recombinant random **peptide** approach)

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 3 OF 45 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:782072 HCAPLUS
Correction of: 1998:8042

DOCUMENT NUMBER: 135:286176
Correction of: 128:152115

TITLE: **Charge** distribution of flanking **amino acids** inhibits O-glycosylation of several single-site acceptors in vivo

AUTHOR(S): Nehrke, Keith; ten Hagen, Kelly G.; Hagen, Fred K.; Tabak, Lawrence A.

CORPORATE SOURCE: Departments of Dental Research and Biochemistry, School of Medicine and Dentistry, University of Rochester, Rochester, NY, 14642, USA

SOURCE: Glycobiology (1997), 7(8), 1053-1060
CODEN: GLYCE3; ISSN: 0959-6658
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB From surveys of known O-glycosylation sites and in vitro glycosylation assays with synthetic peptide acceptors, it appears that the presence of **charged amino acids** near serine/threonine residues reduces the likelihood of O-glycosylation by UDP-GalNAc polypeptide:N-acetylgalactosaminyl-transferases (ppGaNTases). Previously, the authors demonstrated that the in vivo O-glycosylation of a sequence derived from a known glycosylation site of human von Willebrand factor (PHMAQVTVGPGL) was markedly reduced when charged residues were substituted at position -1 and +3 relative to the single threonine. In contrast, acidic residues at positions -2, +1, and +2 had no effect (Nehrke et al., 1996), suggesting that charge distribution but not charge d. was important. To det. whether the charge distribution effect on O-glycosylation is limited to a specific sequence context or restricted to unique isoforms of ppGaNTase, the authors have analyzed the in vivo O-glycosylation of six secreted recombinant reporter proteins in three different cell backgrounds. The differential presence of known ppGaNTase transcripts was detd. in each cell type by Northern blot anal. Each reporter, which contains a single site of O-glycosylation, was O-glycosylated in a cell-background-specific manner; digestion with O-glycanase and .alpha.-N-acetylgalactosaminidase following mild acid hydrolysis suggested that simple type II core structures were acquired. However, in COS7 cells, one reporter peptide acquired glycosaminoglycans in preference to mucin-type O-glycans. Regardless of cell background or the reporter examd., the substitution of glutamic acid residues at positions -1 and +3 markedly diminished the level of mucin-type O-glycosylation. Charge distribution would appear, therefore, to play a more general role in detg. the extent to which solitary O-glycosylation sites are modified.

CC 13-2 (Mammalian Biochemistry)
Section cross-reference(s): 7

IT Oligosaccharides, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(O-linked; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Glycosylation
Plasmid vectors
Post-translational processing
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Glycosaminoglycans, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Glycoproteins, general, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Fusion proteins (chimeric proteins)

RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
(contg. single glycosylation site; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Structure-activity relationship
(enzyme substrate; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Protein motifs
(glycosylation site; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT 9075-15-4, UDP-GalNAc polypeptide:N-acetylgalactosaminyl-transferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT 202647-27-6 **202647-30-1 202647-33-4**
202647-36-7 202647-38-9 202647-40-3
202647-42-5 202647-44-7 202647-46-9 202647-48-1
202647-50-5 202647-52-7 202647-54-9
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(glycosylation site; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

L34 ANSWER 4 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:690343 HCAPLUS
DOCUMENT NUMBER: 136:6333
TITLE: Systematic Modulation of Michael-Type Reactivity of Thiols through the Use of **Charged Amino Acids**
AUTHOR(S): Lutolf, M. P.; Tirelli, N.; Cerritelli, S.; Colussi, L.; Hubbell, J. A.
CORPORATE SOURCE: Department of Materials and Institute for Biomedical Engineering, ETH Zurich and the University of Zurich, Zurich, Switz.
SOURCE: Bioconjugate Chemistry (2001), 12(6), 1051-1056
CODEN: BCCHES; ISSN: 1043-1802
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A quant. structure-reactivity relationship for the Michael-type addn. of thiols onto acrylates was detd. Several thiol-contg. peptides were investigated by examg. the correlation between the second-order rate const. of their addn. onto PEG-diacrylate and the pKa of the thiols within a peptide. By introducing **charged amino acids** in close proximity to a cysteine, the pKa of the thiol was systematically modulated by electrostatic interactions. Pos. charges from the amino acid arginine decreased the pKa of the thiol and accelerated the reaction with acrylates while neg. charges from aspartic acids showed the opposite effect. A linear correlation between thiolate concns. and kinetic consts. was found, confirming the role of thiolates as the reactive species in this Michael-type reaction. The relevant factors influencing the reactivity were the sign and the no. of the neighboring charges, while the position of these charges had little effect on reactivity. These results provide a basis for the rational design of peptides, where the kinetics,

and thus, selectivity of protein/peptide conjugation with polymeric structures via Michael-type addn. reactions can be controlled.

CC 34-3 (Amino Acids, Peptides, and Proteins)
Section cross-reference(s): 22

ST acrylate Michael addn **peptide** thiol reactivity relationship structure

IT Thiols (organic), reactions
RL: PRP (Properties); RCT (Reactant); RACT (Reactant or reagent)
(peptidyl; quant. relationship between the rate const. of the Michael addn. of a **peptide** thiol onto PEG-diacrylate and the pKa of the thiol within the **peptide**)

IT Michael reaction
Molecular structure-property relationship
Reaction kinetics
(quant. relationship between the rate const. of the Michael addn. of a **peptide** thiol onto PEG-diacrylate and the pKa of the thiol within the **peptide**)

IT **Peptides**, reactions
RL: PRP (Properties); RCT (Reactant); RACT (Reactant or reagent)
(thiols; quant. relationship between the rate const. of the Michael addn. of a **peptide** thiol onto PEG-diacrylate and the pKa of the thiol within the **peptide**)

IT 52-90-4, L-Cysteine, reactions
RL: PRP (Properties); RCT (Reactant); RACT (Reactant or reagent)
(quant. relationship between the rate const. of the Michael addn. of a **peptide** thiol onto PEG-diacrylate and the pKa of the thiol within the **peptide**)

IT **376646-27-4P 376646-28-5P 376646-29-6P**
376646-30-9P 376646-31-0P 376646-32-1P
376646-33-2P 376646-34-3P 376646-35-4P
RL: PRP (Properties); RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(quant. relationship between the rate const. of the Michael addn. of a **peptide** thiol onto PEG-diacrylate and the pKa of the thiol within the **peptide**)

IT 26570-48-9P, Polyethyleneglycol diacrylate
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(quant. relationship between the rate const. of the Michael addn. of a **peptide** thiol onto PEG-diacrylate and the pKa of the thiol within the **peptide**)

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 5 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:382948 HCAPLUS
DOCUMENT NUMBER: 133:331151
TITLE: Approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**
AUTHOR(S): Shimazaki, K.; Uji, K.; Tazume, T.; Kumura, H.; Shimo-Oka, T.
CORPORATE SOURCE: Dairy Science Laboratory, Faculty of Agriculture, Hokkaido University, Sapporo, 060-8589, Japan
SOURCE: International Congress Series (2000), 1195(Lactoferrin: Structure, Function and Applications), 37-46
CODEN: EXMDA4; ISSN: 0531-5131
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal

LANGUAGE: English

AB The affinity of lactoferrin for heparin is one its well-known properties. Certain consensus **sequences** have been proposed for other heparin-binding proteins, such as BBXB, BBBXXB or BXXBBXB, where B denotes a pos. **charged amino acid** residue. The purpose of the present study was to identify the essential amino acid side chain groups of the lactoferrin mol. contributing to the interaction with heparin. The heparin-interacting properties of transferrin family proteins were compared by examg. the heparin-binding activity of various peptides prepd. by chem. synthesis. Each peptide was composed of 10-15 amino acid residues and was synthesized from Fmoc amino acid active esters on a pre-activated cellulose membrane using the SPOTs system. Each of the peptides was incubated with heparin. To detect heparin-interaction, human vitronectin and alk. phosphatase-conjugated anti-vitronectin monoclonal antibody were used. Peptides corresponding to partial **sequences** of human, bovine, pig and goat lactoferrins, human transferrin, chicken ovotransferrin and human melanotransferrin were studied. The results obtained were as follows: of the two BXBB **sequences** in the bovine lactoferrin N-lobe, KCRR (18-21) and RMKK (25-28), the latter was found to be essential for interaction with heparin; the BXBB **sequence** in the C-lobe did not interact with heparin; BXBB and BBXB **sequences** in human transferrin showed no interaction with heparin. These results were consistent with findings obtained in affinity chromatog. expts. using an immobilized heparin column.

CC 6-3 (General Biochemistry)

IT Molecular association
(approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT Lactoferrins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT Protein motifs
(heparin-binding site; approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT Structure-activity relationship
(heparin-binding; approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT Transferrins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(melanotransferrins, **peptide** fragments, heparin-binding activity; approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT Conalbumins
Transferrins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(**peptide** fragments, heparin-binding activity; approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT 9005-49-6, Heparin, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

IT **183623-03-2** 226944-40-7 304640-76-4 304640-77-5
304640-78-6 304640-79-7 304640-80-0 304640-81-1 304640-82-2
304640-83-3 304640-84-4 304640-85-5 304640-86-6 304640-87-7
304640-88-8 304640-89-9 304640-90-2 **304640-91-3**
304640-92-4 304640-93-5 304640-94-6
304640-95-7 304640-96-8 304640-97-9
304640-98-0 304640-99-1 304641-00-7 304641-01-8 304641-02-9
304641-03-0 **304641-04-1 304641-05-2**
304641-06-3 304641-07-4 304641-08-5
304641-09-6 304641-10-9 304641-11-0
304641-12-1 304641-13-2 304641-14-3 304641-15-4 304641-16-5
304641-17-6 304641-18-7 304641-19-8
304641-20-1 304641-21-2 304641-22-3
304641-23-4 304641-24-5 304641-25-6
304641-26-7 304641-27-8 **304641-28-9 304641-29-0**
304641-30-3 304641-31-4 **304641-32-5** 304641-33-6
304641-34-7 304641-35-8 304641-36-9
304641-37-0 304641-38-1 304641-39-2 **304641-40-5**
304641-41-6 304641-42-7
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(heparin binding by model **peptides**; approach to identification and comparison of the heparin-interacting sites of lactoferrin using synthetic **peptides**)

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 6 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:351380 HCAPLUS
DOCUMENT NUMBER: 133:825
TITLE: **Peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity
INVENTOR(S): Collin, Peter Donald
PATENT ASSIGNEE(S): Coastside Bio Resources, USA
SOURCE: PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000029009	A1	20000525	WO 1999-US27289	19991118

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1998-109139P P 19981118
US 1999-157078P P 19991001

OTHER SOURCE(S): MARPAT 133:825

AB A pentapeptide is disclosed having the generic formula A-A-A-B-C (A = nonpolar amino acid; B = polar amino acid; C = **charged amino acid**). In a preferred embodiment, the peptide has the **sequence** A-Pro-Pro-B-C, and in a further preferred

embodiment has the **sequence** of Leu-Pro-Pro-Ser-Arg. In a most preferred embodiment, the peptide comprises at least one D-amino acid. The peptide can be extd. from the epidermis of sea cucumbers. The peptides of the invention are useful for inhibition of tumor progression and/or inflammation in a mammal by administration of 1-5000 mg/kg body wt. The peptide can be administered in conjunction with any suitable carriers or excipients as are known those skilled in the arts via oral delivery forms, e.g. capsules, drinks, powders, rectally via suppositories, or other suitable means.

IC ICM A61K038-00
ICS A61K038-04; C07K005-00; C07K007-00; C07K016-00; C07K017-00

CC 1-12 (Pharmacology)
Section cross-reference(s): 12, 63

ST antitumor antiinflammatory pharmaceutical **peptide** sea cucumber; angiogenesis inhibition **peptide** sea cucumber

IT Disease, animal
(Bechets, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Intestine, disease
(Crohn's, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Disease, animal
(Eales, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Bone, neoplasm
(Ewing's sarcoma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Antitumor agents
(Ewing's sarcoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Histamine receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(H3; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Antitumor agents
(Kaposi's sarcoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Sarcoma
(Kaposi's, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Disease, animal
(Osler-Weber-Rendu, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Bone, disease
(Paget's, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Disease, animal
(Terrien's marginal degeneration, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Chemicals
(burns, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Ion channel blockers
(calcium, L-type; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Artery, disease
(carotid, occlusion, undesired angiogenesis in; **peptides**

having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Burn
(chem., undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Disease, animal
(chronic vitritis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Inflammation
(chronic, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye
(cornea, radial keratotomy, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Transplant and Transplantation
(cornea, rejection, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye
Eye
(cornea, transplant, rejection, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Transplant rejection
(corneal, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Lipids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(degeneration, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye, disease
(diabetic retinopathy, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Joint, anatomical
(discomfort, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Disease, animal
(fibrovascular tissue proliferation, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Blood vessel, neoplasm
Blood vessel, neoplasm
(hemangioma, inhibitors; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Blood vessel, neoplasm
(hemangioma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Antitumor agents
(hemangioma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Neoplasm
(hematol., undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Antitumor agents
(hematol.; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Disease, animal

(hyperviscosity syndrome, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Human herpesvirus
Human herpesvirus 3
Mycobacterium
Protozoa
(infection, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Intestine, disease
(inflammatory; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye, disease
(keratitis, atopic and limbic and pterygium keratitis sicca, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye, disease
(keratoconjunctivitis, epidemic, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye, disease
(keratopathy, corneal neovascularization, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Antitumor agents
(leukemia; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye, disease
(macula, degeneration, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Skin, disease
(marginal keratolysis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Erythema
(multiforme, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Vision
(myopia, and optic pits, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Glaucoma (disease)
(neovascular, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Angiogenesis
Angiogenesis
(neovascularization, eye, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Angiogenesis
(neovascularization, retinal, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Eye, disease
(neovascularization, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Nerve, neoplasm
Nerve, neoplasm

(neuroblastoma, inhibitors; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Nerve, neoplasm
(neuroblastoma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Antitumor agents
(neuroblastoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Artery, disease
Vein
(occlusion, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Bone, neoplasm
Bone, neoplasm
(osteosarcoma, inhibitors; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Bone, neoplasm
(osteosarcoma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Antitumor agents
(osteosarcoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Contact lenses
(overwear, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Disease, animal
(pars planitis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Skin, disease
(pemphigoid, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT **Peptides**, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pentapeptides; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Angiogenesis inhibitors
Anti-inflammatory agents
Antitumor agents
Bronchodilators
Cucumaria frondosa
Immunosuppressants
(**peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT **Peptides**, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Galanin receptors
Interleukin 1.alpha.
Interleukin 6
Tumor necrosis factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Disease, animal

(phylectenulosis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Artery, disease
(polyarteritis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Lasers
(post-laser complications, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Disease, animal
(pseudoxanthoma elasticum, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye, neoplasm
Eye, neoplasm
(retinoblastoma, inhibitors; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye, neoplasm
(retinoblastoma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Antitumor agents
(retinoblastoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye, disease
(retinopathy, detachment, chronic, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye, disease
(retinopathy, neovascularization, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye, disease
(retrolental fibroplasia, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Myoma
(rhabdomyosarcoma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Antitumor agents
(rhabdomyosarcoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Skin, disease
(rosacea, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Antitumor agents
(sarcoma; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye
(sclera, scleritis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Antitumor agents
(solid tumor; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Neoplasm
(solid, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Lupus erythematosus
(systemic, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT Toxoplasma gondii
(toxoplasmosis from, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Injury
(trauma, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Bacteria (Eubacteria)
Fungi
(ulcer, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Intestine, disease
(ulcerative colitis, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT AIDS (disease)
Arthritis
Atherosclerosis
Infection
Leukemia
Lyme disease
Osteoarthritis
Psoriasis
Rheumatoid arthritis
Sarcoidosis
Sickle cell anemia
Sjogren's syndrome
Syphilis
Ulcer
(undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT Eye, disease
(uveitis, chronic, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT 39391-18-9
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(1 and 2; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT 141467-21-2
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(II, calcium/calmodulin-dependent; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT 11103-57-4, Vitamin A
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(deficiency, undesired angiogenesis in; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT 9001-84-7, Phospholipase A2
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pancreatic; **peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)
IT 270078-99-4P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(**peptides** having anticancer, antiinflammatory, and

angiogenesis-inhibiting activity)

IT **111757-56-3**, Lpps **peptide+** 120484-65-3, Lppsr **peptide+**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT 9001-92-7, Protease 9004-06-2, Elastase 71160-24-2, Leukotriene B4 80619-02-9, 5-Lipoxygenase 141436-78-4, Protein kinase C 146480-36-6, Matrix metalloproteinase 9
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

IT 65154-06-5, Platelet-activating factor 119418-04-1, Galanin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**peptides** having anticancer, antiinflammatory, and angiogenesis-inhibiting activity)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 7 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:84832 HCAPLUS
DOCUMENT NUMBER: 132:132335
TITLE: Treatment of autoimmune conditions with copolymer 1 and related copolymers and **peptides**
INVENTOR(S): Aharoni, Rina; Teitelbaum, Dvora; Arnon, Ruth; Sela, Michael; Fridkis-Hareli, Masha; Strominger, Jack L.
PATENT ASSIGNEE(S): Yeda Research and Development Co., Ltd, Israel; President and Fellows of Harvard College
SOURCE: PCT Int. Appl., 147 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000005250	A1	20000203	WO 1999-US16747	19990723
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, US, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9952262	A1	20000214	AU 1999-52262	19990723
EP 1098902	A1	20010516	EP 1999-937423	19990723
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
NO 2001000329	A	20010308	NO 2001-329	20010119
US 2002055466	A1	20020509	US 2001-768872	20010123

PRIORITY APPLN. INFO.:
US 1998-93859P P 19980723
US 1998-101825P P 19980925
US 1998-102960P P 19981002
US 1998-106350P P 19981030

US 1998-108184P P 19981112
US 1999-123675P P 19990309
WO 1999-US16747 W 19990723

AB Polypeptides and peptides are disclosed which contain at least three amino acids randomly joined in a linear array, in which at least one of the three amino acids is an arom. amino acid, at least one of the three amino acids is a **charged amino acid** and at least one amino acid is an aliph. amino acid. In a preferred embodiment, the polypeptide contains three or four of the following amino acids: tyrosine, alanine, glutamic acid or lysine. According to the invention, the polypeptides bind to antigen-presenting cells, purified human lymphocyte antigens (HLA), and/or Copolymer 1-specific T cells. Morever, these polypeptides can be formulated into pharmaceutical compns. for treating autoimmune disease. Also disclosed are methods of treating an autoimmune disease in a mammal by administering a pharmaceutically effective amt. of any one of the polypeptides or peptides.

IC C07K007-00

CC 1-7 (Pharmacology)
Section cross-reference(s): 63

ST polypeptide **peptide** copolymer 1 autoimmune disease; antigen presenting cell binding polypeptide autoimmune disease; HLA antigen binding polypeptide autoimmune disease; T cell binding polypeptide autoimmune disease

IT Nervous system
(Guillain-Barre syndrome; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Structure-activity relationship
(HLA-DR-binding; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR1; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR2; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR4; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(MHC (major histocompatibility complex), class II; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(MHC (major histocompatibility complex); copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Cell activation
Cell proliferation
(T cell; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Myelin basic protein
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(T-cell responsive to; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Cytotoxic agents
(T-cell; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT T cell (lymphocyte)
(activation; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Amino acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(aliph.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Nutrients
(anti-; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)
IT Antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(antigenic **peptides**; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Amino acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(arom.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(autoantigens; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT B cell (lymphocyte)
T cell (lymphocyte)
(autoimmune disease mediated by; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Anemia (disease)
(autoimmune hemolytic anemia; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Ovary, disease
(autoimmune oophoritis; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Thyroid gland, disease
(autoimmune thyroiditis; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Eye, disease
(autoimmune uveoretinitis; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Encephalomyelitis
(autoimmune; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Amino acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cationic, and anionic; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT Intestine, disease

(colitis; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Dermatitis
(contact; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Nucleic acids
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(copeptide-encoding; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Anti-inflammatory agents
Antiarthritics
Antidiabetic agents
Antigen-presenting cell
Antirheumatic agents
Autoimmune disease
Drug delivery systems
Graves' disease
Immunosuppressants
Macrophage
Myasthenia gravis
Psoriasis
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT **Peptides**, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Interleukin 2
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Antibacterial agents
Antiviral agents
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)

IT Antibodies
Cytokines
Interleukin 10
Interleukin 4
Steroids, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)

IT Nerve, disease
(demyelination; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Toxins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(enterotoxin B, staphylococcal; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Toxins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(enterotoxins, staphylococcal A; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(guinea pig basic protein; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Purpura (disease)
(idiopathic thrombocytopenic, chronic; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Enzymes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)

IT Drug delivery systems
(injections, i.m.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Drug delivery systems
(injections, i.v.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Drug delivery systems
(injections, s.c.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Hypothyroidism
(myxedema, idiopathic; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Anti-inflammatory agents
(nonsteroidal; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)

IT Drug delivery systems
(oral; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Skin, disease
(pemphigus vulgaris; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Polyamides, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(poly(amino acids); copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Proliferation inhibition
(proliferation inhibitors, T-cell; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT T cell (lymphocyte)
(proliferation; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Cytokine receptors
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sol.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)

IT Drug delivery systems
(solns., i.p.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Lupus erythematosus
(systemic; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Multiple sclerosis

(therapeutic agents; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Toxins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(toxic shock syndrome 1; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Drug delivery systems
(transdermal; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Collagens, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(type II, **peptide**, T-cell responsive to; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT Interferons
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)

IT 50-67-9, Serotonin, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(RBL cell release; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT **175800-89-2**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(collagen **peptide** CII 261-273; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT 59822-51-4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT 136337-22-9 136337-24-1 256480-31-6 256480-32-7 256480-33-8
256480-34-9 256480-35-0 256480-36-1 256480-37-2 256480-38-3
256480-39-4 256480-40-7 256480-41-8 256480-42-9 256480-43-0
256480-45-2 256480-47-4 256480-49-6 256480-51-0 256480-53-2
256480-55-4 256480-56-5 256480-57-6 **256480-58-7**
256480-59-8 **256480-61-2** 256636-27-8 256636-28-9
256636-29-0 256636-30-3 256636-31-4 256636-32-5 256636-33-6
256636-34-7 256636-35-8 256636-36-9 256636-37-0 256636-38-1
256636-39-2 256636-40-5 256636-41-6 256636-42-7 256636-43-8
256636-44-9 256636-45-0 256636-46-1 256636-47-2 256636-48-3
256636-49-4 256636-50-7 256636-51-8 256636-52-9 256636-53-0
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT 27456-63-9P, Alanine-glutamic acid-lysine copolymer 28675-46-9P
31325-29-8P 217075-83-7P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)

IT 28704-27-0 147245-92-9, Copolymer 1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT 56-84-8, L-Aspartic acid, biological studies 61-90-5, L-Leucine, biological studies 63-91-2, L-Phenylalanine, biological studies 72-18-4, L-Valine, biological studies 73-32-5, L-Isoleucine, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT **122630-93-7**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(influenza virus hemagglutinin fragment 306-318; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT 9001-92-7, Protease 39391-18-9, Cyclooxygenase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease, and use with other agents)
IT 129988-08-5
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(myelin basic protein fragment 84-102; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT 6235-35-4 7220-68-0 20556-11-0 35978-98-4 136337-06-9 175177-15-8 256469-72-4 256469-73-5 256469-74-6
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(**peptide** contg. **sequence** of; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT 56-41-7, L-Alanine, biological studies 56-86-0, L-Glutamic acid, biological studies 56-87-1, L-Lysine, biological studies 60-18-4, L-Tyrosine, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(terpolymer contg.; copolymer 1 and related copolymers and **peptides** for treatment of autoimmune disease)
IT 256480-30-5
RL: PRP (Properties)
(unclaimed **sequence**; treatment of autoimmune conditions with copolymer 1 and related copolymers and **peptides**)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 8 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:84830 HCAPLUS
DOCUMENT NUMBER: 132:136417
TITLE: Synthetic **peptides** and methods of use for autoimmune disease therapies
INVENTOR(S): Strominger, Jack L.; Fridkis-Hareli, Masha
PATENT ASSIGNEE(S): The President and Fellows of Harvard College, USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000005249	A2	20000203	WO 1999-US16617	19990722
WO 2000005249	A3	20001005		
W: AU, CA, IL, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9952234	A1	20000214	AU 1999-52234	19990722
EP 1105414	A2	20010613	EP 1999-937393	19990722
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-93859P P 19980723
US 1999-123675P P 19990309
WO 1999-US16617 W 19990722

AB The invention provides heteropolymer compns. and peptide compns., and methods of making and using therapeutic compns. comprising amino acid heteropolymers for treatment of a subject for an autoimmune or an inflammatory disease, the heteropolymer compns. made by solid state synthesis. The invention also provides kits for assaying binding of a compn. to a water-sol. MHC protein.

IC C07K007-00

CC 15-2 (Immunochemistry)
Section cross-reference(s): 63

IT Nervous system
(Guillain-Barre syndrome; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR1; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR2; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR4; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HLA-DR; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(MHC (major histocompatibility complex), class II; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Protein motifs
(MHC class II-binding; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Amino acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(aliph.; prepn. of synthetic MHC class II-binding heteropolymer

peptides for autoimmune disease therapies)
IT Amino acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(arom.; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Antigens
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(autoantigens; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Ovary, disease
(autoimmune oophoritis; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Thyroid gland, disease
(autoimmune thyroiditis; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Drug delivery systems
(carriers; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Intestine, disease
(colitis; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT T cell (lymphocyte)
(cytotoxic; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Nerve, disease
(demyelination; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Diabetes mellitus
(insulin-dependent; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Hypothyroidism
(myxedema; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT **Amino acids**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(neg. or pos. **charged**; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Skin, disease
(pemphigus vulgaris; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Arthritis
Autoimmune disease
Graves' disease
Inflammation
Multiple sclerosis
Myasthenia gravis
Protein sequences
Psoriasis
Rheumatoid arthritis
Test kits
(prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT Hemagglutinins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)
IT **Peptides**, biological studies
RL: BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic

preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Lupus erythematosus
(systemic; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Purpura (disease)
(thrombocytopenic, chronic immune; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Collagens, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type II; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT Eye, disease
(uveitis; prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT **122630-93-7P** **175800-89-2P** 256480-29-2P 256480-30-5P
256480-31-6P 256480-32-7P 256480-33-8P 256480-34-9P 256480-35-0P
256480-36-1P 256480-37-2P 256480-38-3P 256480-39-4P 256480-40-7P
256480-41-8P 256480-42-9P 256480-43-0P 256480-45-2P 256480-47-4P
256480-49-6P 256480-51-0P 256480-53-2P 256480-55-4P 256480-56-5P
256480-57-6P
RL: BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT 6235-35-4D, heteropolymers 7220-68-0D, heteropolymers 20556-11-0D, heteropolymer 35978-98-4D, heteropolymers 136337-06-9D, heteropolymers 175177-15-8D, heteropolymers 256469-72-4D, heteropolymers 256469-73-5D, heteropolymers **256480-58-7D**, heteropolymers **256480-59-8D**, heteropolymers **256480-60-1D**, heteropolymers **256480-61-2D**, heteropolymers
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(prepn. of synthetic MHC class II-binding heteropolymer **peptides** for autoimmune disease therapies)

IT 256636-43-8 256636-44-9 256636-45-0 256636-46-1 256636-47-2
256636-48-3 256636-49-4 256636-50-7 256636-51-8 256636-52-9
256636-53-0
RL: PRP (Properties)
(unclaimed sequence; synthetic **peptides** and methods of use for autoimmune disease therapies)

L34 ANSWER 9 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:736928 HCAPLUS
DOCUMENT NUMBER: 131:350264
TITLE: Antibodies to dendritic cells and human dendritic cell populations with therapeutic and diagnostic applications involving vaccines and adoptive immunotherapy
INVENTOR(S): Rieber, Ernst Peter
PATENT ASSIGNEE(S): Micromet G.m.b.H., Germany
SOURCE: PCT Int. Appl., 121 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9958678	A2	19991118	WO 1999-EP3218	19990511
WO 9958678	A3	20000210		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2328415	AA	19991118	CA 1999-2328415	19990511
AU 9940399	A1	19991129	AU 1999-40399	19990511
EP 1078060	A2	20010228	EP 1999-923574	19990511
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002514420	T2	20020521	JP 2000-548469	19990511

PRIORITY APPLN. INFO.: EP 1998-108534 A 19980511
WO 1999-EP3218 W 19990511

AB Antibodies specifically recognizing a distinct population of human dendritic cells (DCs) and methods of isolating said DCs using said antibodies. Furthermore, antigens and epitopes recognized by the above-described antibodies as well as polynucleotides encoding said antibodies. Also vectors comprising said polynucleotides as well as host cells transformed therewith and their use in the prodn. of said antibodies. Addnl., polypeptides comprising a domain of the binding site of the aforementioned antibodies, or an antigen or epitope described above and at least one further, preferably functional domain as well as polynucleotides encoding such polypeptides. Furthermore, vectors comprising said polynucleotides, host cells transfected with said polynucleotide or vector and their use for the prepn. of the above-described polypeptides. Further a method for isolating or identifying DCs as defined above as well as DCs obtainable by said method and/or characterized by recognition of the above-described antibody, and/or contg. the aforementioned antigen or epitope. Moreover, a method for prepg. or identifying T cells in a certain status as well as methods for identifying compds. which interfere with T cell mediated activation of immune responses. In addn. kits, and compns., preferably pharmaceutical and diagnostic compns. are provided comprising any of the afore described antibodies, antigens, epitopes, polypeptides, polynucleotides, vectors, dendritic cells or T cells or compds. obtainable by the aforementioned method. Methods for identifying mols. synthesized by dendritic cells having enhancing or modulating or suppressing effects on antigen-activation of T cells by gene expression comparisons. Method for dendrite cell propagation are described assocd. with growth in cytokine cocktail and cell immortalization. Activation of T cells against recall and neoantigens is also described.

IC ICM C12N015-13
ICS C07K016-18; C07K016-46; C12N005-20; C07K014-705; C12N015-86; C12N005-10; C07K019-00; C12N015-62; C12N005-06; G01N033-53; A61K039-00; A61K039-395; A61K031-70; A61K035-14; A01K067-027; G01N033-577; G01N033-68; C12Q001-68; A61K048-00

CC 15-3 (Immunochemistry)
Section cross-reference(s): 3, 6

IT **Amino acids**, biological studies
RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical

study); BIOL (Biological study)
(pos. or neg. **charged**; bound to solid support; antibodies to human dendritic cells with therapeutic and diagnostic applications involving vaccines and adoptive immunotherapy)

IT **168650-46-2**
RL: PRP (Properties)
(unclaimed sequence; antibodies to dendritic cells and human dendritic cell populations with therapeutic and diagnostic applications involving vaccines and adoptive immunotherapy)

L34 ANSWER 10 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:672987 HCAPLUS
DOCUMENT NUMBER: 131:307670
TITLE: Methods and compositions for high yield production of eukaryotic proteins in a bacterial cell
INVENTOR(S): Breyer, Richard M.; Ma, Lijun; Kennedy, Chris
PATENT ASSIGNEE(S): Vanderbilt University, USA
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9953033	A1	19991021	WO 1999-US8214	19990416
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9936444	A1	19991101	AU 1999-36444	19990416
US 6383777	B1	20020507	US 1999-293170	19990416
PRIORITY APPLN. INFO.:			US 1998-81989P P	19980416
			WO 1999-US8214 W	19990416

AB The present invention provides nucleic acid constructs comprising a first nucleotide **sequence** encoding a leader **sequence** of DNA binding protein, such as a bacteriophage lambda repressor cI, positioned upstream and in frame with a second nucleotide **sequence** encoding a protein. The leader **sequence** can be between 15 to 76 amino acids long and comprise at least three pos. **charged** **amino acid** residues. A method of producing a eukaryotic protein in a bacterial cell in high yield is also provided, comprising: (a) introducing the expression vector of this invention, wherein the second nucleotide **sequence** encodes a eukaryotic protein, into the bacterial cell; and (b) culturing the bacterial cell under conditions whereby the second nucleotide **sequence** of the expression vector is expressed to produce the eukaryotic protein in high yield is also provided. Eukaryotic proteins can include, but are not limited to the group consisting of integral membrane proteins, G-protein coupled receptor proteins and ion channel proteins.

IC ICM C12N005-16
ICS C12N015-33; C12N015-62; C12N015-63
CC 3-2 (Biochemical Genetics)
Section cross-reference(s): 6, 10, 16
ST eukaryotic protein high yield prodn bacterial cell; repressor cI leader **peptide** integral membrane protein prodn; ion channel protein cI leader **peptide** prodn bacterial cell; G protein coupled receptor cI leader **peptide** bacteria prodn
IT Prostanoid receptors
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified);

THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(EP2, human PG-E2, fusion protein with repressor cI signal **peptide**, prodn. of; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Protein **sequences**
(for plasmid vectors pLJM5.22H, pCK2.5HTL, pSD1.63his, pSD1.18his, pSD1.134his, pSD2.46his, pLJM6-09, pLJM5-42T; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT G protein-coupled receptors
Ion channel
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fusion protein with repressor cI signal **peptide**, prodn. of; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Rho protein (G protein)
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human small, fusion protein with repressor cI signal **peptide**, prodn. of; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Proteins, specific or class
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(membrane, integral, eukaryotic, fusion protein with repressor cI signal **peptide**, prodn. of; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Signal **peptides**
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(of cI protein, coding **sequence** for; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT DNA **sequences**
(of plasmid vectors pLJM5.22H, pCK2.5HTL, pSD1.63his, pSD1.18his, pSD1.134his, pSD2.46his, pLJM6-09, pLJM5-42T; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Potassium channel
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(rat renal outer medullary, fusion protein with repressor cI signal **peptide**, prodn. of; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Chemokine receptors
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(.beta. chemokine receptor CCR5, human, fusion protein with repressor cI signal **peptide**; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT Adrenoceptors
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(.beta.2, human, fusion protein with repressor cI signal **peptide**, prodn. of; methods and compns. for high yield prodn.

of eukaryotic proteins in bacterial cell)

IT 247144-90-7P 247144-91-8P 247195-35-3P 247195-36-4P 247195-37-5P
247195-38-6P 247195-39-7P 247195-40-0P 247195-41-1P 247195-42-2P
247195-43-3P 247195-44-4P 247195-45-5P 247195-46-6P
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid **sequence**; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT **158734-08-8**
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(amino acid **sequence**; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT 247164-28-9, DNA (synthetic plasmid vector pLJM5.22H) 247164-29-0, DNA (synthetic plasmid vector pCK2.5HTL) 247164-30-3 247164-31-4 247164-32-5 247164-33-6 247164-34-7 247164-54-1, DNA (synthetic plasmid vector pLJM6-09) 247164-55-2, DNA (synthetic plasmid vector pLJM5-42T) 247164-56-3, DNA (coliphage .lambda. gene cI) 247164-57-4 247164-58-5 247164-59-6 247164-60-9
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process); USES (Uses)
(nucleotide **sequence**; methods and compns. for high yield prodn. of eukaryotic proteins in bacterial cell)

IT 247196-67-4, PN: WO9953033 SEQID: 16 unclaimed DNA 247196-68-5, PN: WO9953033 SEQID: 17 unclaimed DNA 247196-69-6, PN: WO9953033 SEQID: 18 unclaimed DNA 247196-70-9, PN: WO9953033 SEQID: 19 unclaimed DNA 247196-71-0, PN: WO9953033 SEQID: 20 unclaimed DNA 247196-72-1, PN: WO9953033 SEQID: 21 unclaimed DNA 247196-76-5, PN: WO9953033 SEQID: 22 unclaimed DNA 247196-79-8, PN: WO9953033 SEQID: 23 unclaimed DNA 247196-80-1, PN: WO9953033 SEQID: 24 unclaimed DNA 247196-81-2, PN: WO9953033 SEQID: 25 unclaimed DNA 247196-82-3, PN: WO9953033 SEQID: 26 unclaimed DNA 247196-83-4, PN: WO9953033 SEQID: 27 unclaimed DNA 247196-84-5, PN: WO9953033 SEQID: 28 unclaimed DNA 247196-85-6, PN: WO9953033 SEQID: 29 unclaimed DNA 247196-86-7, PN: WO9953033 SEQID: 30 unclaimed DNA 247196-87-8, PN: WO9953033 SEQID: 31 unclaimed DNA 247196-91-4, PN: WO9953033 SEQID: 32 unclaimed DNA 247196-92-5, PN: WO9953033 SEQID: 33 unclaimed DNA 247196-93-6, PN: WO9953033 SEQID: 34 unclaimed DNA 247196-94-7, PN: WO9953033 SEQID: 35 unclaimed DNA 247196-95-8, PN: WO9953033 SEQID: 36 unclaimed DNA 247196-96-9, PN: WO9953033 SEQID: 37 unclaimed DNA
RL: PRP (Properties)
(unclaimed nucleotide **sequence**; methods and compns. for high yield prodn. of eukaryotic proteins in a bacterial cell)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 11 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:475232 HCAPLUS
DOCUMENT NUMBER: 131:252091
TITLE: A method for computational combinatorial **peptide** design of inhibitors of Ras protein
AUTHOR(S): Zeng, Jun; Treutlein, Herbert R.
CORPORATE SOURCE: Ludwig Institute for Cancer Research, Royal Melbourne Hospital, Parkville, 3050, Australia
SOURCE: Protein Engineering (1999), 12(6), 457-468
CODEN: PRENE9; ISSN: 0269-2139
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A computational combinatorial approach is proposed for the design of a peptide inhibitor of Ras protein. The procedure involves three steps. First, a "Multiple Copy Simultaneous Search" identifies the location of specific functional groups on the Ras surface. This search method allowed the authors to identify an important binding surface consisting of two .beta. strands (residues 5-8 and 52-56), in addn. to the well known Ras effector loop and switch II region. The two .beta. strands had not previously been reported to be involved in Ras-Raf interaction. Second, after constructing the peptide inhibitor chain based on the location of N-methylacetamide (NMA) min., functional groups are selected and connected to the main chain C.alpha. atom. This step generates a no. of possible peptides with different **sequences** on the Ras surface. Third, potential inhibitors are designed based on a **sequence** alignment of the peptides generated in the second step. This computational approach reproduces the conserved pattern of hydrophobic, hydrophilic and **charged amino acids** identified from the Ras effectors. The advantages and limitations of this approach are discussed.

CC 1-3 (Pharmacology)

ST computational combinatorial **peptide** design Ras protein inhibitor

IT Structure-activity relationship
(Ras protein-inhibiting; a method for computational combinatorial **peptide** design of inhibitors of Ras protein)

IT Computer application
Conformation
Drug design
Hydrogen bond
Molecular association
Peptide library
(a method for computational combinatorial **peptide** design of inhibitors of Ras protein)

IT **Peptides**, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(a method for computational combinatorial **peptide** design of inhibitors of Ras protein)

IT Ras proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(a method for computational combinatorial **peptide** design of inhibitors of Ras protein)

IT Chemistry
(computational; a method for computational combinatorial **peptide** design of inhibitors of Ras protein)

IT 244776-94-1 244776-95-2 244776-96-3 244776-97-4 244776-98-5
244776-99-6 244777-00-2 244777-01-3 244777-02-4 244777-04-6
244777-05-7 244777-06-8 244777-07-9 244777-08-0 244777-09-1
244777-10-4 **244777-11-5**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(a method for computational combinatorial **peptide** design of inhibitors of Ras protein)

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 12 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:34494 HCAPLUS
DOCUMENT NUMBER: 130:91268
TITLE: An aggregation-resistant linker **peptide** for

single-chain antibodies and other fusion proteins
INVENTOR(S): Whitlow, Marc D.; Filpula, David R.
PATENT ASSIGNEE(S): Enzon, Inc., USA
SOURCE: U.S., 39 pp., Cont.-in-part of U.S. Ser. No. 2,845, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5856456	A	19990105	US 1994-224591	19940407
US 5990275	A	19991123	US 1997-926789	19970910
PRIORITY APPLN. INFO.:			US 1992-980529	19921120
			US 1993-2845	19930115
			US 1994-224591	19940407

OTHER SOURCE(S): MARPAT 130:91268

AB A linker peptide that can be used in the construction of single-chain antibodies and other fusion proteins is described. This peptide provides greater stability and is less susceptible to aggregation than previously known peptide linkers. The linker may be up to about 50 amino acids long and contains at least one occurrence of a **charged amino acid** followed by a proline. When used for making a single chain antibodies, the linker is preferably from 18 to about 30 amino acids in length.

IC ICM C12N015-11
ICS C12N015-62; C12N015-13; C07H021-04
NCL 536023400
CC 3-2 (Biochemical Genetics)
Section cross-reference(s): 6, 15
ST linker **peptide** fusion protein single chain antibody
IT Fusion proteins (chimeric proteins)
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT Chimeric gene
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(for Ig family fusion proteins; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT cDNA **sequences**
(for single chain antibodies; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT Immunoglobulins
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(fusion products, linker **peptides** for; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT TCR (T cell receptors)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(fusion products,linker **peptides** for; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(heavy chains, fusion proteins contg.; aggregation-resistant linker

peptide for single-chain antibodies and other fusion proteins)
IT Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(light chains, fusion proteins contg.; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT **Peptides**, biological studies
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(linker **peptides**; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT Protein **sequences**
(of single chain antibodies; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT Aggregation
(**peptides** resistant to; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT Antibodies
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(single chain; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT 150067-51-9P 150067-53-1P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation)
(amino acid **sequence**; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT 219481-21-7 219481-23-9
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid **sequence**; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT 130838-28-7 **150243-58-6** 150243-59-7 **150243-60-0**
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(linker **peptide** for single-chain antibody; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT 153177-60-7 157960-67-3 158113-53-2
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(linker **peptide**; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT 56-87-1, L-Lysine, biological studies 74-79-3, L-Arginine, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(linker **peptides** contg.; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
IT 150067-50-8 150067-52-0 219481-22-8 219481-24-0
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide **sequence**; aggregation-resistant linker **peptide** for single-chain antibodies and other fusion proteins)
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 13 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:700283 HCAPLUS
DOCUMENT NUMBER: 130:34916
TITLE: Definition and redesign of the extended substrate

specificity of granzyme B
AUTHOR(S): Harris, Jennifer L.; Peterson, Erin P.; Hudig, Dorothy; Thornberry, Nancy A.; Craik, Charles S.
CORPORATE SOURCE: Department of Pharmaceutical Chemistry, University of California, San Francisco, CA, 94143, USA
SOURCE: Journal of Biological Chemistry (1998), 273(42), 27364-27373
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Granzyme B is a protease involved in the induction of rapid target cell death by cytotoxic lymphocytes. Definition of the substrate specificity of granzyme B allows for the identification of in vivo substrates in this process. By using the combinatorial methods of synthetic substrate libraries and substrate-phage display, an optimal substrate for granzyme B that spans over six subsites was detd. to be Ile-Glu-Xaa-(Asp .dwnarw. Xaa)-Gly, with cleavage of the Asp .dwnarw. Xaa peptide bond. Granzyme B proteolysis was shown to be highly dependent on the length and **sequence** of the substrate, supporting the role of granzyme B as a regulatory protease. Arginine 192 was identified as a determinant of P3-Glu and P1-Asp substrate specificity. Mutagenesis of arginine 192 to glutamate reversed the preference for neg. **charged amino acids** at P3 to pos. **charged amino acids**. The preferred substrate **sequence** matches the activation sites of caspase 3 and caspase 7 and thus is consistent with the role of granzyme B in activation of these proteases during apoptosis. The caspase substrate poly(ADP)-ribose polymerase is cleaved by granzyme B in a cell-free assay at two sites that resemble the granzyme B specificity detd. by the combinatorial methods. Many caspase substrates contain granzyme B cleavage sites and are proposed as potential granzyme B targets, suggesting a redundant function with certain caspases.

CC 7-3 (Enzymes)

ST granzyme B **peptide** specificity combinatorial library; caspase activation substrate granzyme B

IT **Peptides**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(definition and redesign of the extended substrate specificity of granzyme B)

IT 41149-01-3 70967-97-4 **70968-04-6 72682-69-0 72682-73-6** 102838-95-9 108929-37-9 131068-47-8 143180-74-9D, Granzyme B, complexes with optimal substrate **peptide** 165174-58-3 216757-29-8 216757-30-1 216757-31-2 216757-32-3 216757-33-4 216757-34-5 216757-35-6 216757-36-7 **216757-37-8 216757-38-9 216757-39-0D**, complexes with granzyme B
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(definition and redesign of the extended substrate specificity of granzyme B)

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 14 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:380524 HCAPLUS
DOCUMENT NUMBER: 129:146042
TITLE: Enhanced efficiency of a targeted fusogenic **peptide**
AUTHOR(S): Decout, A.; Labeur, C.; Goethals, M.; Brasseur, R.;

Vandekerckhove, J.; Rosseneu, M.
CORPORATE SOURCE: Laboratory for Lipoprotein Chemistry, Department of Biochemistry, Universiteit Gent, Ghent, B-9000, Belg.
SOURCE: Biochimica et Biophysica Acta (1998), 1372(1), 102-116
CODEN: BBACAQ; ISSN: 0006-3002
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Membrane targeting was investigated as a potential strategy to increase the fusogenic activity of an isolated fusion peptide. This was achieved by coupling the fusogenic carboxy-terminal part of the .beta.-amyloid peptide (A.beta., amino acids 29-40), involved in Alzheimer's disease, to a pos. charged peptide (PIP2-binding peptide, PBP) interacting specifically with a naturally occurring neg. charged phospholipid, phosphatidylinositol 4,5-bisphosphate (PIP2). Peptide-induced vesicle fusion was spectroscopically evidenced by: (i) mixing of membrane lipids, (ii) mixing of aq. vesicular contents, and (iii) an irreversible increase in vesicle size, at concns. five to six times lower than the A.beta.(29-40) peptide. In contrast, at these concns., the PBP-A.beta.(29-40) peptide did not display any significant activity on neutral vesicles, indicating that neg. charged phospholipids included as targets in the membranes, are required to compensate for the lower hydrophobicity of this peptide. When the .alpha.-helical structure of the chimeric peptide was induced by dissolving it in trifluoroethanol, an increase of the fusogenic potential of the peptide was obsd., supporting the hypothesis that the .alpha.-helical conformation of the peptide is crucial to trigger the lipid-peptide interaction. The specificity of the interaction between PIP2 and the PBP moiety, was shown by the less efficient targeting of the chimeric peptide to membranes charged with phosphatidylserine. These data thus demonstrate that the specific properties of both the A.beta.(29-40) and the PBP peptides are conserved in the chimeric peptide, and that a synergetic effect is reached through chem. linkage of these two fragments.
CC 6-6 (General Biochemistry)
ST membrane fusion fusogenic **peptide** alpha helix; phosphatidylinositol bisphosphate binding **peptide** beta amyloid
IT Fusion proteins (chimeric proteins)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); PROC (Process)
(PBP-A.beta.(29-40); enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)
IT **Peptides**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); RCT (Reactant); BIOL (Biological study); PROC (Process); RACT (Reactant or reagent)
(PBP; enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)
IT Membrane, biological
(bilayer; enhanced membrane fusion efficiency of targeted **peptide** composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)
IT Phosphatidylinositol 4,5-bisphosphate
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

(Biological study); PROC (Process)
(enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

IT Fusion, biological
(enhanced membrane fusion efficiency of targeted **peptide** composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

IT .alpha.-Helix
(role in fusogenic potential; enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

IT Amyloid
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(.beta.-; enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. charged phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

IT 210841-25-1P
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); PROC (Process)
(**amino acid** sequence of; enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. **charged** phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

IT **184865-04-1**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(**amino acid** sequence of; enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. **charged** phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

IT 210841-26-2
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); RCT (Reactant); BIOL (Biological study); PROC (Process); RACT (Reactant or reagent)
(**amino acid** sequence of; enhanced membrane fusion efficiency of targeted **peptide** PBP-A.beta.(29-40) composed of fusogenic C-terminal part of .beta.-amyloid **peptide** and pos. **charged** phosphatidylinositol 4,5-bisphosphate-binding **peptide** PBP)

L34 ANSWER 15 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:28180 HCAPLUS
DOCUMENT NUMBER: 128:150757
TITLE: Selection of phage display combinatorial library **peptides** with affinity for a yohimbine imprinted methacrylate polymer
AUTHOR(S): Berglund, Johanna; Lindbladh, Christer; Mosbach, Klaus; Nicholls, Ian A.

CORPORATE SOURCE: Department of Pure and Applied Biochemistry, University of Lund, Lund, S-221 00, Swed.
SOURCE: Analytical Communications (1998), 35(1), 3-7
CODEN: ANCOFE; ISSN: 1359-7337
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English

AB .alpha.2-Adrenoreceptor mimics, prepd. by mol. imprinting of yohimbine, were used to select ligands from a phage display hexapeptide library. Phages with affinity for the yohimbine imprinted methacrylic acid-ethylene glycol dimethacrylate copolymer were selected. Phage affinities were estd. using an enzyme immunoassay. The selected library showed three-fold higher affinity for the imprinted polymer compared with the primary library. Eighty-two of ninety characterized phage clones from the selected library showed low affinity for the polymer. The hexapeptides on eight of these low binding phage clones consisted of mainly hydrophobic amino acid residues, and four clones were identical. The hexapeptides on five of the eight high affinity phage clones contained pos. **charged amino acids**. Identical hexapeptides were expressed on four of these five clones. The results of this study suggest that the majority of the selected phages form hydrophobic and/or ionic interactions with the polymer framework rather than specific interactions only with the yohimbine imprinted sites. Furthermore, a direct correlation can be seen between hexapeptide **sequence** affinity and the no. of pos. **charged amino acid** residues they contain.

CC 6-3 (General Biochemistry)
ST phage **peptide** library yohimbine methacrylate polymer
IT **Peptide** library
Phage display library
Protein **sequences**
(selection of phage display combinatorial library **peptides** with affinity for a yohimbine imprinted methacrylate polymer)
IT 202460-77-3 202460-78-4 **202460-79-5 202460-80-8** **202460-81-9** 202460-82-0
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(selection of phage display combinatorial library **peptides** with affinity for a yohimbine imprinted methacrylate polymer)
IT 202537-22-2P 202537-23-3P
RL: BUU (Biological use, unclassified); PRP (Properties); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); USES (Uses)
(selection of phage display combinatorial library **peptides** with affinity for a yohimbine imprinted methacrylate polymer)

L34 ANSWER 16 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:15770 HCAPLUS
DOCUMENT NUMBER: 128:74293
TITLE: T cell antigen receptor **peptides**
INVENTOR(S): Manolios, Nicholas
PATENT ASSIGNEE(S): Northern Sydney Area Health Services, Australia; Manolios, Nicholas
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9747644 A1 19971218 WO 1997-AU367 19970611
W: AU, CA, JP, US
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
CA 2257973 AA 19971218 CA 1997-2257973 19970611
AU 9730193 A1 19980107 AU 1997-30193 19970611
AU 739130 B2 20011004
EP 960119 A1 19991201 EP 1997-924813 19970611
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2000512282 T2 20000919 JP 1998-500998 19970611
PRIORITY APPLN. INFO.: AU 1996-389 A 19960611
AU 1996-390 A 19960611
AU 1996-391 A 19960611
AU 1996-392 A 19960611
AU 1996-393 A 19960611
AU 1996-394 A 19960611
WO 1997-AU367 W 19970611

AB The present invention provides peptides which affect T-cells, presumably by action on the T-cell antigen receptor. The present invention further relates to the therapy of various inflammatory and autoimmune disease states involving the use of these peptides. Specifically, the peptides are useful in the treatment of disorders where T-cells are involved or recruited. In one aspect the peptides have the formula: R1-A-B-A-R2 in which A is a hydrophobic amino acid or a hydrophobic peptide **sequence** comprising between 2 and 10 amino acids; B is a **charged amino acid**; R1 is NH2 and R2 is COOH.
In another aspect the peptides have the formula: R1-A-B-C-R2 in which A is a peptide **sequence** of between 0 and 5 amino acids; B is cysteine; C is a peptide **sequence** of between 2 to 10 amino acids; R1 is NH2; and R2 is COOH.

IC ICM C07K004-00
ICS C07K007-06; C07K014-00; A61K038-08; A61K047-48
CC 15-2 (Immunochemistry)
ST T cell antigen receptor **peptide** inflammation; autoimmune disease
T cell antigen receptor

IT Animal cell
Autoimmune disease
Drug targeting
Inflammation
Protein **sequences**
T cell (lymphocyte)
(T cell antigen receptor **peptides** for treating inflammation and autoimmune diseases)

IT TCR (T cell receptors)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(T cell antigen receptor **peptides** for treating inflammation and autoimmune diseases)

IT Disease, animal
(T cell-involved; T cell antigen receptor **peptides** for treating inflammation and autoimmune diseases)

IT **200556-60-1P**
RL: SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(0T cell antigen receptor **peptides** for treating inflammation and autoimmune diseases)

IT **180994-66-5P 186487-61-6P 186487-64-9P**
186487-70-7P 200556-42-9P **200556-46-3P**
200556-50-9P 200556-51-0P 200556-52-1P
200556-53-2P **200556-54-3P 200556-56-5P**
200556-58-7P 200556-59-8P

RL: SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(T cell antigen receptor **peptides** for treating inflammation and autoimmune diseases)

L34 ANSWER 17 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:8042 HCAPLUS
DOCUMENT NUMBER: 128:152115
TITLE: **Charge** distribution of flanking **amino acids** inhibits O-glycosylation of several single-site acceptors in vivo
AUTHOR(S): Nehrke, Keith; Hagen, Kelly G. Ten; Hagen, Fred K.; Tabak, Lawrence A.
CORPORATE SOURCE: Departments of Dental Research and Biochemistry, School of Medicine and Dentistry, University of Rochester, Rochester, NY, 14642, USA
SOURCE: Glycobiology (1997), 7(8), 1053-1060
CODEN: GLYCE3; ISSN: 0959-6658
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB From surveys of known O-glycosylation sites and in vitro glycosylation assays with synthetic peptide acceptors, it appears that the presence of **charged amino acids** near serine/threonine residues reduces the likelihood of O-glycosylation by UDP-GalNAc polypeptide:N-acetylgalactosaminyl-transferases (ppGaNTases). Previously, the authors demonstrated that the in vivo O-glycosylation of a sequence derived from a known glycosylation site of human von Willebrand factor (PHMAQVTVGPGL) was markedly reduced when charged residues were substituted at position -1 and +3 relative to the single threonine. In contrast, acidic residues at positions -2, +1, and +2 had no effect (Nehrke et al., 1996), suggesting that charge distribution but not charge d. was important. To det. whether the charge distribution effect on O-glycosylation is limited to a specific sequence context or restricted to unique isoforms of ppGaNTase, the authors have analyzed the in vivo O-glycosylation of six secreted recombinant reporter proteins in three different cell backgrounds. The differential presence of known ppGaNTase transcripts was detd. in each cell type by Northern blot anal. Each reporter, which contains a single site of O-glycosylation, was O-glycosylated in a cell-background-specific manner; digestion with O-glycanase and .alpha.-N-acetylgalactosaminidase following mild acid hydrolysis suggested that simple type II core structures were acquired. However, in COS7 cells, one reporter peptide acquired glycosaminoglycans in preference to mucin-type O-glycans. Regardless of cell background or the reporter examd., the substitution of glutamic acid residues at positions -1 and +3 markedly diminished the level of mucin-type O-glycosylation. Charge distribution would appear, therefore, to play a more general role in detg. the extent to which solitary O-glycosylation sites are modified.

CC 13-2 (Mammalian Biochemistry)
Section cross-reference(s): 7

IT Oligosaccharides, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(O-linked; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

IT Glycosylation
(biol.; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of

several single-site acceptors in vivo)
IT Plasmid vectors
Post-translational processing
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT Glycosaminoglycans, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT Glycoproteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT Fusion proteins (chimeric proteins)
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
(contg. single glycosylation site; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT Structure-activity relationship
(enzyme substrate; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT Protein motifs
(glycosylation site; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT 9075-15-4, UDP-GalNAc polypeptide:N-acetylgalactosaminyl-transferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)
IT 202647-27-6 **202647-30-1 202647-33-4**
202647-36-7 202647-38-9 202647-40-3
202647-42-5 202647-44-7 202647-46-9 202647-48-1
202647-50-5 202647-52-7 202647-54-9
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(glycosylation site; **charge** distribution of flanking **amino acids** of protein glycosylation site inhibits O-glycosylation of several single-site acceptors in vivo)

L34 ANSWER 18 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:682652 HCAPLUS
DOCUMENT NUMBER: 127:315957
TITLE: Intramolecular Assistance of cis/trans Isomerization of the Histidine-Proline Moiety
AUTHOR(S): Reimer, Ulf; El Mokdad, Nasr; Schutkowski, Mike; Fischer, Gunter
CORPORATE SOURCE: Enzymology of Protein Folding Research Unit, Halle/Saale, D-06120, Germany
SOURCE: Biochemistry (1997), 36(45), 13802-13808
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Peptidyl-prolyl cis/trans isomerization is a slow conformational interconversion in the polypeptide backbone that is frequently rate-limiting in refolding of proteins and is thought to play a role in cellular restructuring of proteins. In order to probe the influence of pos. **charged amino acids** located in **sequence** segments adjacent to proline, the rotational barriers of Arg-Pro- and His-Pro-contg. peptides were detd. by isomer-specific proteolysis and dynamic NMR spectroscopy for Suc-Ala-His-Pro-Phe-NH-Np, Ac-Ala-Arg-Pro-Ala-Lys-NH2, Ac-Ala-His-Pro-Ala-Lys-NH2, angiotensin III, TSH-releasing hormone (TRH), and [His(3-Me)2]TRH in aq. soln. In contrast to the guanidinium group of arginine, the protonated side chain of histidine preceding proline led to an acceleration of the prolyl isomerization up to 10-fold relative to the unprotonated state. Both arginine and histidine residues succeeding proline in an amino acid **sequence** proved to be ineffective. Under basic and acidic conditions, the kinetic solvent deuterium isotope effects k2c.fwdarw.tHO/k2c.fwdarw.tDO for angiotensin III were 1.0 .+-. 0.1 and 2.0 .+-. 0.1, resp. The results are interpreted in terms of intramol. general acid catalysis of prolyl bond rotation by the imidazolium group that is without precedent in intermol. catalysis.
CC 6-3 (General Biochemistry)
IT Structure-activity relationship
(cis/trans isomerization-affecting, of **peptide**; intramol. assistance by protonated histidine (but not arginine) of cis/trans isomerization of **peptide** proline moiety)
IT Conformation (protein)
Protonation
cis-trans Isomerization
(intramol. assistance by protonated histidine (but not arginine) of cis/trans isomerization of **peptide** proline moiety)
IT 71-00-1, L-Histidine, biological studies 74-79-3, L-Arginine, biological studies
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(intramol. assistance by protonated histidine (but not arginine) of cis/trans isomerization of **peptide** proline moiety)
IT 147-85-3, L-Proline, biological studies
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(intramol. assistance by protonated histidine (but not arginine) of cis/trans isomerization of **peptide** proline moiety)
IT 12687-51-3, Angiotensin III 24305-27-9, Thyrotropin-releasing hormone 34367-54-9 128802-74-4 128802-75-5 194670-58-1 194670-61-6 **197726-99-1**
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(**peptide**; intramol. assistance by protonated histidine (but not arginine) of cis/trans isomerization of **peptide** proline moiety)

L34 ANSWER 19 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:623434 HCAPLUS
DOCUMENT NUMBER: 127:330064
TITLE: TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin
AUTHOR(S): Spee, Pieter; Neefjes, Jacques
CORPORATE SOURCE: Department Cellular Biochemistry, Netherlands Cancer

Institute, Amsterdam, 1066 CX, Neth.
SOURCE: Eur. J. Immunol. (1997), 27(9), 2441-2449
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The endoplasmic reticulum (ER) membrane-embedded transporter assocd. with antigen processing (TAP) assocs. with peptides in the cytosol and translocates these into the ER lumen. MHC I mols. bind a subset of these peptides and the remainder is either removed or degraded, or may be retained in the ER in assocn. with other proteins. Peptide-binding proteins were visualized in the ER using radioactive peptides with a photoreactive group. Besides TAP, 2 proteins were identified as gp96 and protein disulfide isomerase (PDI). Calreticulin, previously found in complex with TAP, only binds glycosylated peptides. In addn., 2 as yet unidentified, ER luminal glycoproteins (gp120 and gp170) were visualized. The effects of peptide size and **sequence** on binding to the ER-resident proteins were studied by partially degenerated peptides with photoreactive side chains. All identified proteins were able to bind peptides within the size range of peptides translocated by TAP, from 8 to >20 amino acids. Whereas PDI assocd. with all peptides tested, gp96 and gp120 showed a clear **sequence** preference for non-**charged** **amino acids** at positions 2 and 9 in 9mer peptides. Thus various ER proteins, other than the MHC class I heterodimer and TAP, are able to interact with peptide albeit with a different substrate selectivity.

CC 15-2 (Immunochemistry)
Section cross-reference(s): 6

ST **peptide** binding protein TAP endoplasmic reticulum; transporter antigen processing **peptide** endoplasmic reticulum

IT Antigen processing
Endoplasmic reticulum
Intracellular transport
Peptide-binding structure-activity relationship
(TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT Antigens
Calreticulin
Class I MHC antigens
Peptides, biological studies
TAP-1 (transporter in antigen processing 1)
TAP-2 (transporter in antigen processing 2)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT Glycoproteins (specific proteins and subclasses)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(gp120; TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT Glycoproteins (specific proteins and subclasses)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(gp170; TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT Glycoproteins (specific proteins and subclasses)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(gp61; TAP-translocated **peptides** specifically bind proteins

in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT Glycoproteins (specific proteins and subclasses)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(gp96; TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT 37318-49-3, Protein disulfide isomerase
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

IT **158442-97-8D**, radioiodinated derivs. 197894-34-1D, radioiodinated derivs. 197894-35-2D, radioiodinated derivs. 197894-36-3D, radioiodinated derivs.
RL: BPR (Biological process); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(TAP-translocated **peptides** specifically bind proteins in the endoplasmic reticulum, including gp96, protein disulfide isomerase, and calreticulin)

L34 ANSWER 20 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:407869 HCAPLUS
DOCUMENT NUMBER: 127:148107
TITLE: Use of eluted **peptide sequence** data to identify the binding characteristics of **peptides** to the insulin-dependent diabetes susceptibility allele HLA-DQ8 (DQ 3.2)
AUTHOR(S): Godkin, Andrew; Friede, Thomas; Davenport, Miles; Stevanovic, Stefan; Willis, Anthony; Jewell, Derek; Hill, Adrian; Rammensee, Hans-Georg
CORPORATE SOURCE: Mol. Immunology Group, Inst. Mol. Medicine, Gastroenterology Unit, John Radcliffe Hospital, Oxford, OX3 9DU, UK
SOURCE: International Immunology (1997), 9(6), 905-911
CODEN: INIMEN; ISSN: 0953-8178
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB HLA-DQ8 (A1*0301, B1*0302) and -DQ2 (A1*0501, B1*0201) are both assocd. with diseases such as insulin-dependent diabetes mellitus and celiac disease. The authors used the technique of pool sequencing to look at the requirements of peptides binding to HLA-DQ8, and combined these data with naturally **sequenced** ligands and in vitro binding assays to describe a novel motif for HLA-DQ8. The motif, which has the same basic format as many HLA-DR mols., consists of four or five anchor regions, in the positions from the N-terminus of the binding core of n, n + 3, n + 5/6 and n + 8, i.e. P1, P4, P6/7 and P9. P1 and P9 require neg. or polar residues, with mainly aliph. residues at P4 and P6/7. The features of the HLA-DQ8 motif were then compared to a pool **sequence** of peptides eluted from HLA-DQ2. A consensus motif for the binding of a common peptide which may be involved in disease pathogenesis is described. Neither of the disease-assocd. alleles HLA-DQ2 and -DQ8 have Asp at position 57 of the .beta.-chain. This Asp, if present, may form a salt bridge with an Arg at position 79 of the .alpha.-chain and so alter the binding specificity of P9. HLA-DQ2 and -DQ8 both appear to prefer neg. **charged amino acids** at P9. In contrast, HLA-DQ7 (A1*0301, B1*0301), which is not assocd. with diabetes, has Asp at .beta.57, allowing pos. **charged amino acids** at P9. This anal. of the **sequence** features of DQ-binding

peptides suggests mol. characteristics which may be useful to predict epitopes involved in disease pathogenesis.
CC 15-2 (Immunochemistry)
ST **peptide** motif HLA DQ8
IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(HLA-DQ2; **peptide** motif for binding to HLA-DQ8 and)
IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(HLA-DQ8; **peptide** motif for binding to HLA-DQ8)
IT Diabetes mellitus
(insulin-dependent; **peptide** motif for binding to HLA-DQ8 in relation to)
IT Amino acids, biological studies
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(of **peptide** motif for binding to HLA-DQ8)
IT Protein motifs
(**peptide** motif for binding to HLA-DQ8)
IT **Peptides**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(**peptide** motif for binding to HLA-DQ8)
IT 193343-12-3 **193343-13-4** 193343-14-5 193343-15-6
193343-16-7 **193343-17-8** 193343-18-9
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(**peptide** motif for binding to HLA-DQ8)

L34 ANSWER 21 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:218866 HCAPLUS
DOCUMENT NUMBER: 126:304176
TITLE: Effect of size and charge on the passive diffusion of **peptides** across Caco-2 cell monolayers via the paracellular pathway
AUTHOR(S): Pauletti, Giovanni M.; Okumu, Franklin W.; Borchardt, Ronald T.
CORPORATE SOURCE: Dep. Pharmaceutical Chem., Univ. Kansas, Lawrence, KS, 66047, USA
SOURCE: Pharm. Res. (1997), 14(2), 164-168
CODEN: PHREEB; ISSN: 0724-8741
PUBLISHER: Plenum
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of size and charge on the permeation characteristics of peptides across the intestinal mucosa was evaluated. The lipophilicities of neutral, pos. and neg. charged capped amino acids (Asn, Lys, Asp), tripeptides (Ac-Gly-X-Ala-NH2; X = Asn, Lys, Asp) and hexapeptides (Ac-Trp-Ala-Gly-Cly-X-Ala-NH2; X = Asn, Lys, Asp) were estd. using an immobilized artificial membrane. The diffusion coeffs. used to calc. the mol. radii were measured by NMR. The transport characteristics of the model peptides were detd. across Caco-2 cell monolayers. When model compds. having the same charge were compared, permeation was highly size-dependent (capped amino acids > tripeptides . hexapeptides), suggesting transport predominantly via the paracellular route. For example, the flux of the neg. charged Asp amino acid (Papp = 10.04 .+-. 0.43 .times. 10-8 cm/s) was 3 times greater than that obsd. for the

Asp-contg. hexapeptide (Papp = 3.19 .+-. 0.27 .times. 10-8 cm/s). When model compds. of the same size were compared, permeation across the cell monolayer was charge-dependent (neg. < pos. .ltoreq. neutral). For example, the neutral, Asn-contg. tripeptide (Papp = 25.79 .+-. 4.86 .times. 10-8 cm/s) was substantially more able to permeate the Caco-2 cell monolayer than the neg. charged Asp-contg. tripeptide (Papp = 7.95 .+-. 1.03 .times. 10-8 cm/s) and the pos. charged Lys-contg. tripeptide (Papp = 9.86 .+-. 0.18 .times. 10-8 cm/s). The permeability of the cell monolayer to peptides became less sensitive to net charge as the size of the peptides increased. A pos. net charge of hydrophilic peptides enhances their permeation across the intestinal mucosa via the paracellular pathway. With increasing mol. size, mol. sieving of the epithelial barrier dominates the transport of peptides, and the effect of the net charge becomes less significant.

CC 13-6 (Mammalian Biochemistry)

ST **peptide** permeability intestine mucosa size charge

IT **Amino acids**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(capped; effect of size and **charge** on passive diffusion of **peptides** across intestinal mucosal cell monolayers via the paracellular pathway)

IT Intestinal mucosa
Permeation (biological)
(effect of size and charge on passive diffusion of **peptides** across intestinal mucosal cell monolayers via the paracellular pathway)

IT Tripeptides
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(effect of size and charge on passive diffusion of **peptides** across intestinal mucosal cell monolayers via the paracellular pathway)

IT **Peptides**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(hexapeptides; effect of size and charge on passive diffusion of **peptides** across intestinal mucosal cell monolayers via the paracellular pathway)

IT 19789-60-7 60803-67-0 84652-30-2 189232-60-8 **189232-61-9**
189232-62-0 **189232-63-1** 189232-64-2 189232-65-3
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(effect of size and charge on passive diffusion of **peptides** across intestinal mucosal cell monolayers via the paracellular pathway)

L34 ANSWER 22 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:24223 HCAPLUS
DOCUMENT NUMBER: 126:88053
TITLE: Binding of **peptides** naturally presented by HLA-B27 to the differentially disease-associated B*2704 and B*2706 subtypes, and to mutants mimicking their polymorphism
AUTHOR(S): Galocha, B.; Lamas, J. R.; Villadangos, J. A.; Albar, J. P.; De Castro, J. A. Lopez
CORPORATE SOURCE: Centro de Biologia Molecular Severo Ochoa (C.S.I.C.-U.A.M.), Facultad de Ciencias, Universidad Autonoma de Madrid, Madrid, Spain
SOURCE: Tissue Antigens (1996), 48(5), 509-518
CODEN: TSANA2; ISSN: 0001-2815
PUBLISHER: Munksgaard
DOCUMENT TYPE: Journal
LANGUAGE: English

AB B*2704 and B*2706 are closely related HLA-B27 subtypes of which the former but not the latter is assocd: to ankylosing spondylitis. Their peptide specificity relative to other disease-assocd. subtypes was analyzed by

testing binding of self-peptides naturally presented by B*2705 or B*2702, and synthetic analogs, to B*2704, B*2706, and site-specific mutants mimicking their changes. Peptides with basic, aliph. or arom. C-terminal residues bound to B*2705 with similar affinity. In B*2704 C-terminal aliph./arom. residues were preferred. B*2706 discriminated drastically between polar and nonpolar C-terminal residues, showing strong preference for Leu and Phe, and less than B*2704 for basic and Tyr residues. Loss of single acidic charges (D>S77, D>Y116) increased preference for C-terminal Leu and Phe, but allowed efficient binding of peptides with basic residues or Tyr. Their gain (V>E152, H>D114) maintained wide C-terminal specificity, but severely impaired binding, presumably by disrupting interactions with internal peptide residues. This was compensated by Y116 in the double D114Y116 mutant. The specificity of B*2704 and B*2706 was explained only partially by the sep. effects of single mutations, indicating that novel properties arise from concomitant changes at various positions. For instance, specificity of B*2706 for nonpolar C-terminal residues required simultaneous removal of Asp77 and Asp116. B*2706 differed from B*2705, B*2702, and B*2704 in its lower suitability for C-terminal Tyr, suggesting that this feature might be relevant for HLA-B27 assocn. to spondyloarthropathy.

CC 15-2 (Immunochemistry)

ST **peptide** specificity HLA B27 allele spondyloarthropathy

IT Genes (animal)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HLA-B*2704; modulation of **peptide** binding by HLA-B27 polymorphism in relation to)

IT Genes (animal)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HLA-B*2706; modulation of **peptide** binding by HLA-B27 polymorphism in relation to)

IT Structure-activity relationship
(histocompatibility antigen HLA-B27-binding; of **peptides**)

IT Amino acids, biological studies
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(in modulation of **peptide** binding by HLA-B27 polymorphism)

IT HLA-B27 antigen
Peptides, biological studies
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(modulation of **peptide** binding by HLA-B27 polymorphism)

IT Alleles
Ankylosing spondylitis
Susceptibility (genetic)
(modulation of **peptide** binding by HLA-B27 polymorphism in relation to)

IT Electric **charge** (biological)
(of **amino acids** in relation to modulation of **peptide** binding by HLA-B27 polymorphism)

IT 137532-68-4 137532-69-5 137532-70-8 137532-71-9 137532-72-0
137532-73-1 137532-74-2 137532-75-3 137532-76-4 137532-77-5
137532-78-6 155352-90-2 155353-03-0 157896-44-1 183202-02-0
183202-03-1 185611-54-5 185611-55-6 185611-56-7 185611-57-8
185611-58-9 185611-59-0 185611-60-3 **185611-61-4**
185611-62-5 185611-63-6 185611-64-7 185611-65-8 185611-66-9
185611-67-0 185611-68-1 185611-69-2 185611-70-5 185611-71-6
185611-72-7 185611-73-8 185611-74-9 185611-75-0 185611-76-1
185611-77-2 185611-78-3
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)

(modulation of **peptide** binding by HLA-B27 polymorphism)

L34 ANSWER 23 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:842649 HCAPLUS
DOCUMENT NUMBER: 123:246823
TITLE: Hydrophilic signal oligopeptides and methods of therapeutic use
INVENTOR(S): Rath, Matthias
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9519568	A1	19950720	WO 1995-US575	19950112
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9516810	A1	19950801	AU 1995-16810	19950112
EP 744027	A1	19961127	EP 1995-908522	19950112
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
AU 9881834	A1	19981008	AU 1998-81834	19980824
AU 735298	B2	20010705		

PRIORITY APPLN. INFO.: US 1994-182248 A 19940114
WO 1995-US575 W 19950112

AB The instant invention is directed to a method of identifying signal oligopeptides through the use of algorithms, the use of signal oligopeptides as vaccines and as immunogens to produce antibodies. Like the human language, the protein code consists of letters, words, and sentences. The letters (amino acids) and sentences (complete 3-dimensional proteins) have been known previously, but the present discovery identifies the protein words or verbs. These protein verbs are represented by signal oligopeptides which are localized on the surface of the protein and are represented by the hydrophilicity maxima of the protein. These signal oligopeptides are enriched in **charged amino acids** in a versatile arrangement with neutral spacer amino acids. The sp. signal character of these oligopeptides is detd. by a characteristic combination of conformation and charge within the signal **sequence**. Sas in human language, the whole sentence (complete 3-dimensional protein) is needed to det. the sp. and complete action of any given protein. In human language eliminating or changing the verb of a sentence renders the whole sentence meaningless. Similarly, blocking the protein code verbs (signal oligopeptides) can be therapeutically used to block the undesired action or interaction of an entire protein. The discovery of the protein code provides the rationale for deciphering the communication code of diseases. Infectious diseases, cancer, cardiovascular and other diseases develop by means of one or more pathogenicity-mediating protein. Blocking the signal oligopeptides of these proteins (e.g., with antibodies) allows the sp. therapeutic interception of a pathol. communication and thereby blocks disease propagation. Some 360 oligopeptides of signal significance are presented.

IC ICM G01N033-531

CC 1-7 (Pharmacology)
Section cross-reference(s): 6, 7, 15
ST hydrophilic signal oligopeptide code **sequence** therapy; antibody signal oligopeptide **sequence** therapy
IT Algorithm
(for signal **peptide** searching; hydrophilic signal oligopeptides and methods of therapeutic use)
IT **Peptides**, biological studies
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oligo-, hydrophilic signal oligopeptides and methods of therapeutic use)
IT 99713-67-4 168690-08-2 168690-09-3 168690-10-6 168690-11-7
168690-12-8 168690-13-9 168690-14-0 168690-15-1 168690-16-2
168690-17-3 168690-18-4 168690-19-5 168690-20-8 168690-21-9
168690-22-0 168690-23-1 168690-24-2 168690-25-3 168690-26-4
168690-27-5 168690-28-6 168690-29-7 168690-30-0 168690-31-1
168690-32-2 168690-33-3 168690-34-4 168690-35-5 168690-36-6
168690-37-7 168690-38-8 168690-39-9 168690-40-2 168690-41-3
168690-42-4 168690-43-5 168690-44-6 168690-45-7 168690-46-8
168690-47-9 168690-48-0 168690-49-1 168690-50-4 168690-51-5
168690-52-6 168690-53-7 168690-54-8 168690-55-9 168690-56-0
168690-57-1 168690-58-2 168690-59-3 168690-60-6 168690-61-7
168690-62-8 168690-63-9 168690-64-0 168690-65-1 168690-66-2
168690-67-3 168690-68-4 168690-69-5 168690-70-8 168690-71-9
168690-72-0 168690-73-1 168690-74-2 168690-75-3 168690-76-4
168690-77-5 168690-78-6 168690-79-7 168690-80-0 **168690-81-1**
168690-82-2 168690-83-3 168690-84-4 168690-85-5 168690-86-6
168690-87-7 168690-88-8 168690-89-9 **168690-90-2**
168690-91-3 168690-92-4 168690-93-5 168690-94-6 168690-95-7
168690-96-8 168690-97-9 168690-98-0 168690-99-1 168691-00-7
168691-01-8 168691-02-9 168691-03-0 168691-04-1 168691-05-2
168691-06-3 168691-07-4 168691-08-5 168691-09-6 168691-10-9
168691-11-0 168691-12-1 168691-13-2 168691-14-3 168691-15-4
168691-16-5 168691-17-6 168691-18-7 168691-19-8 168691-20-1
168691-21-2 168691-22-3 168691-23-4 168691-24-5 168691-25-6
168691-26-7 168691-27-8 168691-28-9 168691-29-0 168691-30-3
168691-31-4 168691-32-5 168691-33-6 168691-34-7 168691-35-8
168691-36-9 168691-37-0 168691-38-1 168691-39-2 168691-40-5
168691-41-6 168691-42-7 168691-43-8 168691-44-9
168691-45-0 168691-46-1 168691-47-2 **168691-48-3**,
1-8-Gastrin-14 I (human) **168691-49-4** **168691-50-7**
168691-51-8 **168691-52-9** 168691-53-0
168691-54-1 168691-55-2 168691-56-3 168691-57-4
168691-58-5 168691-59-6 168691-60-9 168691-61-0 168691-62-1
168691-63-2 168691-64-3 168691-65-4 168691-66-5 168691-67-6
168691-68-7 168691-69-8 168691-70-1 168691-71-2 168691-72-3
168691-73-4 168691-74-5 168691-75-6 168691-76-7 168691-77-8
168691-78-9 168691-79-0 **168691-80-3** 168691-81-4
168691-82-5 168691-83-6 168691-84-7 168691-85-8 168691-86-9
168691-87-0 168691-88-1 168691-89-2 168691-90-5 168691-91-6
168691-92-7 168691-93-8 168691-94-9 **168691-95-0**
168691-96-1 168691-97-2 168691-98-3 168691-99-4 168692-00-0
168692-01-1 168692-02-2 168692-03-3 168692-04-4 168692-05-5
168692-06-6 168692-07-7 168692-08-8 168692-09-9 168692-10-2
168692-11-3 168692-12-4 168692-13-5 168692-14-6 168692-15-7
168692-16-8 168692-17-9 168692-18-0 **168692-19-1**
168692-20-4 168692-21-5 168692-22-6 168692-23-7 168692-24-8
168692-25-9 168692-26-0 168692-27-1 168692-28-2 168692-29-3
168692-30-6 168692-31-7 168692-32-8 168692-33-9 168692-34-0

168692-35-1 168692-36-2 168692-37-3 168692-38-4 168692-39-5
168692-40-8 168692-41-9
RL: BOC (Biological occurrence); PRP (Properties); THU (Therapeutic use);
BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(hydrophilic signal oligopeptides and methods of therapeutic use)

IT 168692-42-0 168692-43-1 168692-44-2 168692-45-3 168692-46-4
168692-47-5 168692-48-6 168692-49-7 168692-50-0 168692-51-1
168692-52-2 168692-53-3 168692-54-4 168692-55-5 168692-56-6
168692-57-7 168692-58-8 168692-59-9 **168692-60-2**
168692-61-3 168692-62-4 168692-63-5 168692-64-6 168692-65-7
168692-66-8 168692-67-9 168692-68-0 168692-69-1 168692-70-4
168692-71-5 168692-72-6 168692-73-7 168692-74-8 168692-75-9
168692-76-0 168692-77-1 168692-78-2 168692-79-3 168692-80-6
168692-81-7 168692-82-8 168692-83-9 **168692-84-0**
168692-85-1 168692-86-2 168692-87-3 168692-88-4 168692-89-5
168692-90-8 168692-91-9 168692-92-0 168692-93-1 **168692-94-2**
168692-95-3 168692-96-4 168692-97-5 168692-98-6 168692-99-7
168693-00-3 168693-01-4 168693-02-5 168693-03-6 168693-04-7
168693-05-8 168693-06-9 168693-07-0 **168693-08-1**
168693-09-2 168693-10-5 168693-11-6 168693-12-7 168693-13-8
168693-14-9 168693-15-0 168693-16-1 168693-17-2 168693-18-3
168693-19-4 168693-20-7 168693-21-8 168693-22-9 168693-23-0
168693-24-1 168693-25-2 168693-26-3 168693-27-4 168693-28-5
168693-29-6 168693-30-9 168693-31-0 168693-32-1 168693-33-2
168693-34-3 168693-35-4 168693-36-5 168693-37-6 168693-38-7
168693-39-8 168693-40-1 168693-41-2 168693-42-3 168693-43-4
168693-44-5 168693-45-6 168693-46-7 168693-47-8 168693-48-9
168693-49-0 168693-50-3 168693-51-4 168693-52-5 168693-53-6
168693-54-7 168693-55-8 168693-56-9 168693-57-0
RL: BOC (Biological occurrence); PRP (Properties); THU (Therapeutic use);
BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(hydrophilic signal oligopeptides and methods of therapeutic use)

L34 ANSWER 24 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:756273 HCAPLUS
DOCUMENT NUMBER: 123:132863
TITLE: Compounds useful in anti-allergy treatment
INVENTOR(S): Lewin, Ian Victor; Stanworth, Denis Raymond
PATENT ASSIGNEE(S): Peptide Therapeutics Ltd., UK
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9510532	A1	19950420	WO 1994-GB2230	19941011
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2173856	AA	19950420	CA 1994-2173856	19941011
AU 9478200	A1	19950504	AU 1994-78200	19941011
EP 723553	A1	19960731	EP 1994-928979	19941011
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				

JP 09505563 T2 19970603 JP 1994-511473 19941011
PRIORITY APPLN. INFO.: GB 1993-20897 19931011
WO 1994-GB2230 19941011
OTHER SOURCE(S): MARPAT 123:132863

AB Pharmaceutically acceptable compds. of less than 1200 MW for use in the treatment of IgE-mediated allergies comprise a first neg. charged atom or group and a second neg. charged atom or group, sepd. by a spacing group effective conformationally to position said neg. charged atoms or groups so that they will neutralize the lysine residues of the amino acid **sequence** Lys-Thr-Lys at positions 497-499 of the C.epsilon.4 const. domain of cell-bound human IgE, but excluding any compds. already known for this purpose such as DSCG and nedocromil sodium and toxic compds. In a second aspect, the present invention provides peptides (in which the left-hand side represents the N-terminus and the right-hand side the C terminus) R1mXaa1SpXaa2R2n [R1, R2 = residue of amino acid or of **sequence** of 2-3 amino acids (preferably any amino acid residue of R1 or R2 adjacent to an Xaa residue is neither pos. nor neg. charged); Xaa1 = residue of neg. **charged amino acid**, preferably Glu; Sp = spacing residue, preferably of non-**charged amino acid**, preferably Pro, or of non-charged dipeptide, which provides spacing required for the neg. charged groups of the Xaa residues to be sufficiently proximal to the lysine residues of the amino acid **sequence** Lys-Thr-Lys at positions 497-499 of the C.epsilon.4 const. domain of cell-bound human IgE to neutralize them; Xaa2 = residue of a neg. **charged amino acid**, preferably Glu; m, n = no. of amino acids in R1 and R2 resp., and each of m and n independently is 0-3, and m + n = 0-3] and their terminal functional derivs., both per se and for the use in the treatment of IgE-mediated allergies. Peptide Glu-Pro-Glu was tested for anti-allergy activity. When 24 peptides of the invention were tested for effectiveness compared to nedocromil sodium, the peptides showed comparable effectiveness, and in some cases were more effective.

IC ICM C07K005-093
ICS C07K005-10; C07K007-06; A61K038-06; A61K038-07; A61K038-08

CC 1-7 (Pharmacology)

ST allergy inhibitor **peptide**; IgE mediated allergy inhibition **peptide**

IT Allergy inhibitors
(**peptides** for allergy treatment via neutralization of IgE subsequence)

IT **Peptides**, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** for allergy treatment via neutralization of IgE subsequence)

IT Immunoglobulins
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(E, **peptides** for allergy treatment via neutralization of IgE subsequence)

IT 106326-71-0
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(IgE subsequence; **peptides** for allergy treatment via neutralization of IgE subsequence)

IT 107920-12-7 166891-86-7 166891-87-8 166891-88-9 166891-89-0
166891-90-3 166891-91-4 166891-92-5 166891-93-6 166891-94-7
166891-95-8 166891-96-9 166891-97-0 166891-98-1 166891-99-2
166892-00-8 166892-01-9 **166892-02-0** 166892-03-1
166892-04-2 166892-05-3 166892-06-4 166892-07-5 166892-08-6
166892-09-7
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(**peptides** for allergy treatment via neutralization of IgE subsequence)

IT 166891-86-7D, derivs. 166891-87-8D, derivs.
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**peptides** for allergy treatment via neutralization of IgE subsequence)

L34 ANSWER 25 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:478310 HCAPLUS
DOCUMENT NUMBER: 122:256395
TITLE: Cyclic RGD and KGD **peptides** for treating thrombosis
INVENTOR(S): Pierschbacher, Michael D.; Cheng, Soan; Craig, William S.; Tschopp, Juerg F.
PATENT ASSIGNEE(S): La Jolla Cancer Research Foundation, USA
SOURCE: PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9500544	A1	19950105	WO 1994-US6913	19940617
W: AU, BB, BG, BR, BY, CA, CN, CZ, FI, HU, JP, KP, KR, KZ, LK, LV, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SK, UA, US, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6017877	A	20000125	US 1994-246852	19940519
AU 9471127	A1	19950117	AU 1994-71127	19940617
EP 705274	A1	19960410	EP 1994-920270	19940617
EP 705274	B1	20000830		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09501910	T2	19970225	JP 1994-503001	19940617
AT 195949	E	20000915	AT 1994-920270	19940617
US 5672585	A	19970930	US 1995-445745	19950522
US 5780303	A	19980714	US 1995-459566	19950602

PRIORITY APPLN. INFO.:

US 1993-79441	A	19930618
US 1993-171068	A	19931220
US 1990-506444	B2	19900406
US 1991-681119	B1	19910405
US 1993-50736	B2	19930414
US 1994-246852	A1	19940519
WO 1994-US6913	W	19940617

OTHER SOURCE(S): MARPAT 122:256395

AB Cyclic RGD and KGD peptides, synthesized by methods well-known in the art, inhibit platelet aggregation without causing prolonged bleeding time. Typically these peptides contain hydrophobic amino acids adjacent to the carboxy terminus of the RGD or KGD **sequence**. Peptides of the invention can also contain in addn. to the hydrophobic amino acid an adjacent pos. **charged amino acid**. These peptides have a high affinity for the receptor IIb/IIIa and a low affinity for the fibronectin and vitronectin receptors. The peptides can be administered in a suitable physiol. acceptable carrier to therapeutically treat thrombosis.

IC ICM C07K007-06
ICS C07K007-08; C07K007-10; A61K037-02; G01N033-68
CC 1-3 (Pharmacology)
Section cross-reference(s): 34, 63

ST RGD KGD cyclic **peptide** thrombosis treatment

IT Anticoagulants and Antithrombotics
Blood platelet aggregation inhibitors
Molecular structure-biological activity relationship
(cyclic RGD and KGD **peptides** for treating thrombosis)

IT **Peptides**, biological studies
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(cyclic RGD and KGD; cyclic RGD and KGD **peptides** for treating thrombosis)

IT Brain, disease
(stroke, cyclic RGD and KGD **peptides** for treating thrombosis)

IT **91037-75-1P** 126716-28-7P 126716-30-1P 126716-31-2P
138297-06-0P 138297-07-1P 138297-08-2P 138297-09-3P 138297-10-6P
138297-11-7P 138297-12-8P 138297-13-9P 138297-15-1P 138297-16-2P
138297-18-4P 138297-19-5P 138297-20-8P 138297-21-9P 138297-22-0P
138297-28-6P 138297-29-7P 138297-30-0P 138297-31-1P 138297-32-2P
138297-33-3P 138297-34-4P 138297-35-5P 138297-36-6P 138297-38-8P
138297-39-9P 138297-40-2P 138297-41-3P 138297-42-4P 138297-43-5P
138297-44-6P 138297-45-7P 138297-46-8P 138297-47-9P 138297-48-0P
138297-50-4P 138297-51-5P 138297-52-6P 138297-53-7P 138297-55-9P
138297-56-0P 138297-57-1P 138297-58-2P 138297-59-3P 138297-60-6P
138297-61-7P 138297-62-8P 138297-63-9P 138297-64-0P 138297-65-1P
138297-66-2P 138297-67-3P 138297-68-4P 138297-69-5P 138297-70-8P
138297-72-0P 138297-73-1P 138297-74-2P 138297-75-3P 138297-76-4P
138297-77-5P 138297-78-6P 138297-80-0P 138297-81-1P 138297-82-2P
138297-83-3P 138297-84-4P 138297-86-6P 138297-87-7P 138297-88-8P
138297-89-9P 138297-90-2P 138297-91-3P 138297-92-4P 138297-94-6P
138297-95-7P 138297-96-8P 138297-97-9P 138297-98-0P 138318-49-7P
138318-51-1P 138318-52-2P 138318-53-3P 138318-54-4P 138318-56-6P
138318-57-7P 138381-21-2P 153127-92-5P 153127-94-7P 153128-07-5P
153128-08-6P 153128-09-7P 153128-10-0P 153128-11-1P 153128-12-2P
153128-13-3P 153128-15-5P 153128-16-6P 153128-17-7P 161790-78-9P
162096-73-3P 162096-74-4P 162096-75-5P 162096-76-6P 162096-77-7P
162096-78-8P 162096-79-9P 162096-80-2P 162096-81-3P 162096-82-4P
162096-83-5P 162096-84-6P 162096-85-7P 162096-86-8P 162096-87-9P
162096-88-0P 162096-89-1P 162096-90-4P 162096-91-5P 162096-92-6P
162096-93-7P 162096-94-8P 162096-95-9P 162096-96-0P 162096-97-1P
162096-98-2P 162096-99-3P 162097-00-9P 162097-01-0P 162097-02-1P
162097-03-2P 162097-04-3P 162097-05-4P 162097-06-5P 162097-07-6P
162097-08-7P 162097-09-8P 162097-10-1P 162097-11-2P 162097-12-3P
162097-13-4P 162097-14-5P 162097-15-6P 162097-16-7P 162097-17-8P
162097-18-9P 162097-19-0P 162097-20-3P 162097-21-4P 162460-95-9P
162460-96-0P
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(cyclic RGD and KGD **peptides** for treating thrombosis)

L34 ANSWER 26 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:361775 HCAPLUS
DOCUMENT NUMBER: 122:211933
TITLE: Minimal **sequence** requirements for synthetic **peptides** derived from the V3 loop of the human immunodeficiency virus type 1 (HIV-1) to enhance HIV-1 binding to cells and infection
AUTHOR(S): Zanotto, Carlo; Calderazzo, Francesca; Dettin, Monica; Di Bello, Carlo; Autiero, Monica; Guardiola, John; Chieco-Bianchi, Luigi; De Rossi, Anita

CORPORATE SOURCE: Inst. of Oncology, Interuniversity Center for Cancer Research, Padova, Italy
SOURCE: Virology (1995), 206(2), 807-16
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A 23-mer peptide (DB3) derived from the V3 loop of the surface glycoprotein of HIV-1 MN strain is known to bind to sol. CD4 and enhance HIV-1 infection. The mechanism and structural features required for these biol. activities were studied by using shortened DB3 derivs. and DB3 analogs carrying single amino acid substitutions. Peptides in which the arom. amino acid in position 15 or 16 had been replaced by an uncharged hydrophobic residue (DB3-115 and DB3-116), analogs in which pos. **charged amino acids** were replaced by corresponding D-enantiomers, and shortened DB3-derivs. lost both enhancing activity and ability to bind a sol. CD4. Other peptide variants in which a pos. **charged amino acid** was replaced by asparagine at positions 3 (DB3-N3), 6 (DB3-N6), and 19 (DB3-N19), resp., retained both enhancing and binding activities, although with different efficiencies. The CD4 binder peptides DB3 and DB3-N19, but none of the CD4 nonbinder peptides, enhanced CD4 expression on peptide-treated cells as well as gp120 binding to both CD4+ cells and sol. CD4. These findings strongly suggest that the peptide/CD4 interaction induced an increase in both CD4 expression and CD4/gp120 binding affinity, which in turn mediated the enhancement of viral infection. A model of the structural conformation of DB3 peptide required for its biol. activities is discussed.

CC 15-8 (Immunochemistry)

ST HIV1 virus infectivity glycoprotein gp120env **peptide**; CD4 antigen gp120 HIV1 virus **peptide**

IT Conformation and Conformers
(of synthetic **peptides** derived from the V3 loop of HIV-1 virus for enhanced HIV-1 binding to cells and infection)

IT Antigens
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(CD4, minimal **sequence** requirements for synthetic **peptides** derived from the V3 loop of HIV-1 virus to enhance HIV-1 binding to cells and infection)

IT Sialoglycoproteins
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(gp120env, minimal **sequence** requirements for synthetic **peptides** derived from the V3 loop of HIV-1 virus to enhance HIV-1 binding to cells and infection)

IT Virus, animal
(human immunodeficiency 1, minimal **sequence** requirements for synthetic **peptides** derived from the V3 loop of HIV-1 virus to enhance HIV-1 binding to cells and infection)

IT Molecular structure-biological activity relationship
(virus infectivity-enhancing, minimal **sequence** requirements for synthetic **peptides** derived from the V3 loop of HIV-1 virus to enhance HIV-1 binding to cells and infection)

IT 128910-46-3 131473-70-6 **152045-58-4** 161925-60-6
161925-61-7 161925-62-8 161925-63-9 161925-64-0 161925-65-1
161925-66-2 161925-67-3 161925-68-4
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(minimal **sequence** requirements for synthetic **peptides** derived from the V3 loop of HIV-1 virus to enhance HIV-1 binding to cells and infection)

L34 ANSWER 27 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:296191 HCAPLUS
DOCUMENT NUMBER: 120:296191
TITLE: HLA-B27 binding **peptides** derived from the 57 kD heat shock protein of Chlamydia trachomatis: novel insights into the **peptide** binding rules
AUTHOR(S): Daser, Angelika; Urlaub, Henning; Henklein, Peter
CORPORATE SOURCE: Deutsch. Rheumaforschungszent., Berlin, Germany
SOURCE: Mol. Immunol. (1994), 31(5), 331-6
CODEN: MOIMD5; ISSN: 0161-5890
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In this study the authors investigate the 57 kDa heat shock protein of Chlamydia trachomatis for potential HLA-B27 restricted T cell epitopes. This protein is known to elicit T cell immunity, as judged by delayed type hypersensitivity. The authors synthesized 24 peptides contg. the B27 anchor amino acid arginine at position 2, according to the rules previously described for peptide binding to MHC class I mols. The nonamer peptides were tested in an in vitro assembly assay; six out of the 24 peptides bind to HLA-B27 although their **sequences** only partially match the HLA-B27 binding motif. Two of these six peptides carry neg. **charged amino acids** which apparently fit into the P1 pocket and in three out of the six a pos. **charged amino acid** fits into the P3 pocket. In addn., two octamer peptides stabilized the HLA-B27 mol. without contg. an appropriate amino or carboxy terminus. Therefore the authors' data suggest that current binding rules will need to be refined before they can be used to accurately predict potential T cell epitopes. Furthermore the authors' HLA-B27-binding peptides should prove useful probes for the study of the processing and presentation of this bacterial antigen, and of changes in the T cell repertoire induced by this form of infection.

CC 15-2 (Immunochemistry)

IT Chlamydia trachomatis
(57,000-mol.-wt. heat-shock protein of, **peptides** of, HLA-B27 binding of, **peptide** structure in)

IT Molecular structure-biological activity relationship
(histocompatibility antigen HLA-B27-binding, of **peptides** of 57,000-mol.-wt. heat-shock protein of Chlamydia trachomatis)

IT Histocompatibility antigens
RL: BIOL (Biological study)
(HLA-B27, **peptides** of Chlamydia trachomatis 57,000-mol.-wt. heat-shock protein binding to, **peptide** structure in)

IT Lymphocyte
(T-cell, Chlamydia trachomatis 57,000-mol.-wt. heat-shock protein **peptide** structure in HLA-B27 binding in relation to epitopes for)

IT Proteins, specific or class
RL: BIOL (Biological study)
(chaperonins 10, **peptides** of, of Chlamydia trachomatis, HLA-B27 binding of, **peptide** structure in)

IT Proteins, specific or class
RL: BIOL (Biological study)
(chaperonins 60, **peptides** of, of Chlamydia trachomatis, HLA-B27 binding of, **peptide** structure in)

IT 155070-91-0 155070-92-1 155070-93-2 155070-94-3 **155070-95-4**
155070-96-5 155070-97-6 155070-98-7 155070-99-8
155071-00-4 155071-01-5 **155071-02-6** 155071-03-7
155071-04-8 155071-05-9 155071-06-0 155071-07-1 **155071-08-2**
155071-09-3 **155071-10-6** **155071-11-7** 155071-12-8

155071-13-9 155071-14-0
RL: BIOL (Biological study)
(HLA-B27 antigen binding by, of 57,000-mol.-wt. heat-shock protein of Chlamydia trachomatis, structure in)

L34 ANSWER 28 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:214695 HCAPLUS
DOCUMENT NUMBER: 120:214695
TITLE: Identification of conserved T cell receptor CDR3 residues contacting known exposed **peptide** side chains from a major histocompatibility complex class I-bound determinant
AUTHOR(S): Kelly, Janice M.; Sterry, Sandra J.; Cose, Stephen; Turner, Stephen J.; Fecondo, John; Rodda, Stuart; Fink, Pamela J.; Carbone, Francis R.
CORPORATE SOURCE: Dep. Pathol. Immunol., Monash Med. Sch., Prahran, 3181, Australia
SOURCE: Eur. J. Immunol. (1993), 23(12), 3318-26
CODEN: EJIMAF; ISSN: 0014-2980
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have analyzed the T cell receptor (TCR) repertoire found in the major histocompatibility complex class I-restricted cytotoxic T lymphocyte (CTL) response to the protein ovalbumin (OVA). Despite skewing towards the expression of V.beta.5.2+ TCR by OVA-specific CTL from C57BL/6 mice, the authors found a relatively high degree of diversity in V(D)J usage in both TCR .alpha.- and .beta.-chains. Closer examn. showed that the majority of these sequences encoded neg. and pos. charged residues at their resp. TCR .alpha.- and .beta.-chain VJ or VDJ junctions. These junctions form the third complementarity-detg. regions (CDR3) of the TCR polypeptides involved in the direct interaction with the class I-bound peptide. Crystallog. analyses of Kb-peptide complexes predict that the major determinant from OVA, peptide OVA257-264 (SIINFEKL), contains two exposed charged side chains which can contact the TCR. These are the neg. charged glutamic acid at determinant position 6 (P6) and the pos. charged lysine at P7. To examine whether the TCR .alpha.-chain makes contact with P7 lysine, the authors established a single chain TCR transgenic C57BL/6 mouse line where all T cells express a TCR .beta.-chain derived from the V.beta.5.2+ clone B3. OVA-specific T cells derived from in vivo primed transgenic mice preferentially expressed TCR .alpha.-chains that also contained neg. charged junctional residues despite some further variation in V.alpha. and J.alpha. sequences. Stimulation of naive TCR .beta.-chain transgenic T cells with a P7 substitution peptide analog induced a T cell response that was no longer cross-reactive with the wild-type OVA257-264 determinant, suggesting that the TCR .alpha.-chain from the T cell clone B3 can det. the specificity for this residue. Consequently, these results reveal the existence of conserved residues in the CDR3 of TCR .alpha.- and .beta.-chains specific for OVA257-264 and identify their possible orientation over the peptide-class I complex.
CC 15-2 (Immunochemistry)
Section cross-reference(s): 3
ST TCR receptor CDR3 histocompatibility antigen **peptide**; sequence mouse T cell receptor ovalbumin
IT Ovalbumins
RL: BIOL (Biological study)
(antigen receptor of cytotoxic T-cells to, CDR3 residues of, in contact with **peptides** in histocompatibility class I complexes)
IT **Amino acids**, biological studies
RL: BIOL (Biological study)
(**charged**, of CDR3 regions of cytotoxic T-cell antigen

receptors, in binding to **peptides** in histocompatibility class I complexes)

IT Molecular structure-biological activity relationship
(cytotoxic T-cell-stimulating, of antigenic **peptides**, TCR contact residues in)

IT Antigens
RL: BIOL (Biological study)
(**peptides** of, class I histocompatibility complexes of, antigen receptor of cytotoxic T-cells in recognition of, CDR3 contact residues in)

IT Histocompatibility antigens
RL: BIOL (Biological study)
(H-2Kb, complexes, with **peptides**, antigen receptor of cytotoxic T-cells in recognition of, CDR3 contact residues in)

IT Histocompatibility antigens
RL: BIOL (Biological study)
(MHC (major histocompatibility antigen complex), class I, complexes, with **peptides**, antigen receptor of cytotoxic T-cells in recognition of, CDR3 contact residues in)

IT Lymphocyte
(T-cell, cytotoxic, antigen receptor of, CDR3 residues of, in contact with **peptide** in histocompatibility class I complex)

IT Receptors
RL: BIOL (Biological study)
(TCR .alpha..beta. (T-cell antigen receptor .alpha..beta.), CDR3 regions of, contact residues of, in binding to **peptides** in histocompatibility class I complexes)

IT Antigens
RL: BIOL (Biological study)
(TCR .alpha..beta. receptors, CDR3 regions of, contact residues of, in binding to **peptides** in histocompatibility class I complexes)

IT **Peptides**, compounds
RL: BIOL (Biological study)
(complexes, with histocompatibility class I antigens, receptor of cytotoxic T-cells in recognition of, CDR3 contact residues in)

IT **138831-86-4**
RL: BIOL (Biological study)
(of ovalbumin, T-cell antigen receptor to histocompatibility class I-bound, CDR3 contact residues in)

L34 ANSWER 29 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:52437 HCAPLUS
DOCUMENT NUMBER: 120:52437
TITLE: Natural **peptide** ligand motifs of two HLA molecules associated with myasthenia gravis
AUTHOR(S): Malcherek, Georg; Falk, Kirsten; Roetzschke, Olaf; Rammensee, Hans Georg; Stevanovic, Stefan; Gnau, Volker; Jung, Juenther; Melms, Arthur
CORPORATE SOURCE: Neurol. Klin, Univ. Tuebingen, Tuebingen, Germany
SOURCE: Int. Immunol. (1993), 5 1229-37
CODEN: INIMEN; ISSN: 0953-8178
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The peptide motifs of two HLA mols., B8 and DR3(17), which are assocd. with autoimmune diseases including myasthenia gravis, were detd. from natural peptide pools using Edman degrdn. The majority of HLA-B8 ligands are nonamers preferentially terminated by leucine. As a characteristic feature of the HLA-B8 motif, there is a high degree of conservation of pos. **charged amino acids** at position 3 and 5, exclusively lysine at position 3, and lysine or arginine at position 5.

Addnl. evidence for this allele-specific motif is the presence of these features in several viral peptides recognized by HLA-B8 restricted T cells. The DR3(17) motif is characterized by 4 conserved anchor-like positions ordered in an almost sym. arrangement, as has been found for DR1 and DR5 motifs. A first hydrophobic/arom. anchor 3-4 residues apart from the N-terminus (at relative position 1) appears to be a common feature of DR ligands. The second anchor is an aspartate at relative position 4, which is likely to be the DR3(17)-specific contact site in the groove. Two addnl. conserved positions closer to the C-terminus are occupied by **charged amino acids** at relative position 6 and by hydrophobic/arom. residues at positions 8, 9, or 10. Eight individual naturally processed DR17 ligands were **sequenced** and were derived from exogenous proteins and cytoplasmic membrane receptors. These natural peptides conform well to the detd. motif. A single exchange of the anchor-like positions in a model peptide abrogated binding to DR17+ cells. On the basis of the DR17 motif, peptides from the acetylcholine receptor (AChR), the autoimmune target in myasthenia gravis, were selected that should contain candidate epitopes for AChR-specific T helper cells. Several peptide **sequences** which were reported to activate T cells from DR3+ individuals were among this collection.

CC 15-8 (Immunochemistry)
ST **peptide** motif HLA B8 DR3 myasthenia
IT Myasthenia gravis
(natural **peptide** ligand motifs of HLA-B8 and HLA-DR3 antigens assocd. with human)
IT Histocompatibility antigens
RL: BIOL (Biological study)
(HLA-DR3, natural **peptide** ligand motifs of, assocd. with myasthenia gravis in humans)
IT Histocompatibility antigens
RL: BIOL (Biological study)
(HLA-Dw16, natural **peptide** ligand motifs of, assocd. with myasthenia gravis in humans)
IT Lymphocyte
(T-cell, **peptides** recognition by human, HLA-B8 and HLA-DR3 antigen-restricted, myasthenia gravis in relation to)
IT 115521-12-5 132184-62-4 135700-24-2 136013-83-7 138110-79-9
143761-10-8 145151-52-6 146573-73-1 152244-08-1 152244-09-2
152244-10-5 152244-11-6 152244-12-7 152244-13-8 152244-14-9
152244-15-0 152244-16-1 152244-17-2 152244-18-3 152244-19-4
152244-20-7 152244-21-8 152244-22-9 152244-23-0 152244-24-1
152244-25-2 **152244-26-3** 152244-27-4 152244-28-5
152244-29-6 152244-30-9 152244-31-0 152244-32-1 152244-33-2
152244-34-3 152244-35-4 152244-36-5 152244-37-6 152244-38-7
152244-39-8 152244-40-1 152244-41-2 152244-42-3 152244-43-4
152244-44-5 152244-45-6 152244-46-7 152244-47-8 152244-48-9
152244-49-0 152244-50-3 152244-51-4 152244-52-5 152244-53-6
152244-54-7 152244-55-8 152244-56-9 152244-57-0 152244-58-1
152244-59-2 152244-60-5 152244-61-6 152244-62-7 152244-63-8
152244-64-9 152244-65-0 152244-66-1 152244-67-2 152271-71-1
RL: BIOL (Biological study)
(as natural **peptide** ligand motif of HLA-B8 and HLA-DR3 antigens assocd. with myasthenia gravis in humans)

L34 ANSWER 30 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:406748 HCAPLUS
DOCUMENT NUMBER: 119:6748
TITLE: Presentation of endogenous **peptides** to MHC class I-restricted cytotoxic T lymphocytes in transport deletion mutant T2 cells

AUTHOR(S): Zweerink, Hans J.; Gammon, Maureen C.; Utz, Ursula; Sauma, Samir Y.; Harrer, THomas; Hawkins, Julio C.; Johnson, R. Paul; Sirotina, Anna; Hermes, Jeffrey D.; et al.
CORPORATE SOURCE: Dep. Autoimmune Dis. Res., Merck Res. Lab., Rahway, NJ, 07065, USA
SOURCE: J. Immunol. (1993), 150(5), 1763-71
CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The ability of minigene-encoded viral peptide epitopes to be presented by class I mols. in the absence of MHC-encoded transporters was evaluated in mutant T2 cells. These cells have a large deletion in the class II MHC region that includes the known transporter protein for antigenic peptides and proteasome genes and they are defective in presenting viral epitopes to CTL. T2 cells that express minigenes encoding the influenza virus matrix peptide 58-66 (GILGFVFTL) and 2 HTLV 1 Tax peptides 11-19 (LLFGYPVYV) and 12-19 were lysed by HLA-A2-restricted peptide-specific CTL. Minigene expression of a HLA-A2-restricted HIV reverse transcriptase peptide 476-484 (ILKEPVHGV) with 3 charged residues sensitized T2 cells poorly for lysis by HIV-specific CTL unless the peptide was preceded by an endoplasmic reticulum translocation signal **sequence**. Expression of an influenza virus nucleoprotein peptide 383-391 (SRYWAIRTR) with 3 charged arginine residues did sensitize HLA-B27+ T2 cells for lysis by peptide-specific CTL. These and other results with endogeneously expressed peptide analogs in which hydrophobic and **charged amino acids** were interchanged demonstrate that antigenic peptides can be translocated from the cytoplasm into the class I antigen presentation pathway independent of MHC-encoded transporters, and that peptide hydrophobicity appears not to be a major determinant in selecting peptides for this alternate pathway.

CC 15-2 (Immunochemistry)
ST class I antigen **peptide** presentation transporter; T lymphocyte antigen presentation transporter
IT Proteins, specific or class
RL: BIOL (Biological study)
(TAP (transporter in antigen processing), endogenous **peptide** presentation to MHC class I-restricted cytotoxic T-cells independent of, **peptide** hydrophobicity in relation to)
IT Virus, animal
(**peptide** antigens of, presentation to human cytotoxic T-cells of, transporter-independent, hydrophobicity in relation to)
IT 117446-38-5 **139079-41-7 141368-69-6 141677-18-1 141997-16-2** 142479-13-8 147468-95-9 **148138-87-8**
RL: BIOL (Biological study)
(presentation of viral, to class I restricted human T-cells, transporter-independent, hydrophobicity in relation to)

L34 ANSWER 31 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:192247 HCAPLUS
DOCUMENT NUMBER: 118:192247
TITLE: Purification of synthetic **peptides** using reversible chromatographic probes based on the Fmoc molecule
AUTHOR(S): Ball, H. L.; Mascagni, P.
CORPORATE SOURCE: Italfarmaco Res. Cent., Milan, Italy
SOURCE: Int. J. Pept. Protein Res. (1992), 40(5), 370-9
CODEN: IJPPC3; ISSN: 0367-8377
DOCUMENT TYPE: Journal

LANGUAGE: English

AB A rapid, reversible procedure for purifying synthetic peptides has been developed based on the specific incorporation of 9-(4-carboxyfluorenyl)methoxycarbonyl (4-COR-Fmoc; R = lipophilic or charged group) group onto the terminal amino acid of peptidyl resins. The acid-stable 4-COR-Fmoc derivs. were synthesized with a variety of chem. groups, thus altering the chromatog. properties of the target peptides and permitting their convenient purifn., either by reversed-phase HPLC or ion exchange chromatog. The assembly of the peptides involved a capping step to prevent the formation of deletion forms. The 4-COR-Fmoc derivs. were incorporated either as preformed amino acid conjugates or as activated succinimidyl esters. After HF cleavage and purifn., the 4-COR-Fmoc probes were quant. removed with org. bases. The efficiency of the technique was demonstrated by the purifn. of small- to large-sized peptides, including a cyclic analog.

CC 34-3 (Amino Acids, Peptides, and Proteins)

ST carboxyfluorenylmethoxycarbonyl **peptide** chromatog purifn

IT Protective groups
(lipophilic or charged (carboxyfluorenyl)methoxycarbonyl derivs., for purifn. of **peptides**)

IT **Peptides**
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, by solid-phase methods, chromatog. purifn. of (carboxyfluorenyl)methoxycarbonyl derivs. in)

IT Solid phase synthesis
(**peptide**, chromatog. purifn. in, via (carboxyfluorenyl)methoxycarbonyl derivs.)

IT 95303-04-1
RL: RCT (Reactant)
(amidation of, with lipophilic or **charged amino acids** or **peptides**)

IT 146553-30-2P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and acylation by, of resin-bound **peptide**)

IT 146573-63-9P 146573-64-0P 146573-65-1P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and acylation by, of resin-bound **peptides**)

IT **147097-70-9P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. and purifn. and deprotection of, with org. base)

IT 146553-33-5P **146553-34-6P 146553-35-7P** 146553-37-9P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn., purifn., and deprotection of, with org. base)

L34 ANSWER 32 OF 45 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1993:189470 HCAPLUS

DOCUMENT NUMBER: 118:189470

TITLE: Molecular mapping of human band 3 aging antigenic sites and active amino acids using synthetic **peptides**

AUTHOR(S): Kay, Marguerite M. B.

CORPORATE SOURCE: Health Sci. Cent., Univ. Arizona, Tucson, AZ, 85724, USA

SOURCE: J. Protein Chem. (1992), 11(6), 595-602
CODEN: JPCHD2; ISSN: 0277-8033

DOCUMENT TYPE: Journal

LANGUAGE: English

AB An aging antigen, senescent cell antigen appears on old cells and marks them for death by initiating the binding of IgG autoantibody and

subsequent removal by phagocytes. This antigen is derived from the major anion transport protein, protein band 3, that is involved in respiration and acid base balance. Synthetic peptides from the transmembrane, anion transport segment of band 3 were used to identify potential aging antigenic sites. A competitive inhibition assay with affinity purified IgG autoantibody from senescent red cells was used. Results indicate that: aging antigenic sites reside on human band 3 residues 538-554, 593-601, and 812-830; and that the smallest residues which act as aging antigenic sites are 593-601 and 813-818. The contribution of lysine and/or arginine to antigenicity was examd. by synthesizing peptide analogs in which glycines or arginines are substituted for lysines or arginines. Substitution of neutral glycine for the pos. **charged amino acids** arginine or lysine or both arginine and lysine did not result in a difference in antigenicity between the analog and the native band 3 peptide. Substitution of the pos. charged arginine for the pos. charged lysine resulted in a redn. in antigenicity. The chicken **sequence** of band 3 peptides 538-554 and 812-827 differs from that of the human peptides at several sites. Antigenicity of these chicken analogs was tested and compared to the human peptides. Evidently the 3-dimensional configuration of band 3 segments plays a dominant role in defining the antigenic determinants reactive with senescent cell IgG autoantibodies.

CC 15-2 (Immunochemistry)
Section cross-reference(s): 13

IT 130021-09-9 130021-10-2 130021-11-3 130021-12-4 130021-13-5
130021-16-8 130021-17-9 130021-19-1 130021-23-7 130021-27-1
146999-31-7 146999-32-8 **146999-33-9**
146999-34-0 **146999-35-1** **146999-36-2**
146999-37-3 146999-38-4 146999-39-5 146999-40-8 146999-41-9
146999-42-0 146999-43-1 146999-44-2 146999-45-3 146999-46-4
146999-47-5 146999-48-6 146999-49-7 146999-50-0
146999-51-1 146999-52-2 **146999-53-3** **146999-54-4**
146999-55-5 **146999-56-6** 146999-57-7 **146999-58-8**
146999-59-9 146999-60-2 146999-61-3 146999-62-4
RL: BIOL (Biological study)
(senescent cell IgG binding to human band 3 protein inhibition by, structure in relation to)

L34 ANSWER 33 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:610296 HCAPLUS
DOCUMENT NUMBER: 117:210296
TITLE: Identification of a motif for HLA-DR1 binding **peptides** using M13 display libraries
AUTHOR(S): Hammer, Juergen; Takacs, Bela; Sinigaglia, Francesco
CORPORATE SOURCE: Dep. Biol. Pharm. Res. New Technol., F. Hoffmann-La Roche Ltd., Basel, CH-4002, Switz.
SOURCE: J. Exp. Med. (1992), 176(4), 1007-13
CODEN: JEMEAV; ISSN: 0022-1007
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Oligonucleotides encoding peptides known to bind to HLA-DR1 mols. were inserted into the gene III of filamentous M13 phages. DR1 mols. purified from human lymphoblastoid cell lines specifically bound to these peptide **sequences** expressed on the phage surface. A M13 phage peptide library was next constructed and screened with DR1 mols. After 4 rounds of selection, >80% of the phages were able to bind to DR1. Competition expts. with both isolated phages and corresponding synthetic peptides showed that the binding was specific. **Sequence** anal. of the peptide encoding region of 60 phages binding to DR1 mols. and comparison with phages of the original library revealed 2 potential anchor positions.

The first was an arom. residue (Tyr, Phe, or Trp) at the NH2 terminus of the peptide **sequences**, and the second was located 3 residues downstream and consisted of Met or Leu. In addn., the neg. **charged amino acids** Asp and Glu were mostly excluded from the DR1 binding **sequences**, and the small amino acid residues Gly and Ala were enriched at position 6. As for DR1, this approach should make it possible to easily det. the binding motifs of other MHC class II alleles and isotypes.

CC 15-2 (Immunochemistry)
ST HLA DR1 antigen binding **peptide** motif
IT **Peptides**, biological studies
RL: BIOL (Biological study)
(HLA-DR1 antigen-binding, of phage M13 display library)
IT Molecular cloning
(of HLA-DR antigen-binding **peptide**-encoding oligonucleotides, in M13 phage)
IT Protein **sequences**
(of HLA-DR1 antigen-binding **peptides**)
IT Histocompatibility antigens
RL: BIOL (Biological study)
(HLA-DR1, **peptides** binding, motif for, in phage M13 display library)
IT Virus, bacterial
(M13, oligonucleotides encoding HLA-DR antigen-binding **peptides** cloning in)
IT Molecular structure-biological activity relationship
(antigen-binding, HLA-DR1, of recombinant **peptides**)
IT Nucleotides, polymers
RL: PROC (Process)
(oligo-, for HLA-DR1 antigen-encoding **peptides**, mol. cloning of, in M13 phage)
IT **144138-04-5** 144138-05-6 **144138-06-7**
144138-07-8 **144138-08-9** 144138-09-0 144138-10-3
144138-11-4 **144138-12-5** 144138-13-6 144138-14-7
144138-15-8 **144138-16-9** **144138-17-0** 144138-18-1
144138-19-2 **144138-20-5** **144138-21-6**
144138-22-7 144138-23-8 144138-24-9 144138-25-0
144138-26-1 144138-27-2 144138-28-3 **144138-29-4**
144138-30-7 **144138-31-8** **144138-32-9**
144138-33-0 **144138-34-1** **144138-35-2** 144138-36-3
144138-37-4 144138-38-5 144138-39-6 **144138-40-9**
144138-41-0 **144138-42-1** **144138-43-2**
144138-44-3 144138-45-4 144138-46-5 **144138-47-6**
144138-48-7 144138-49-8 **144138-50-1**
144138-51-2 **144138-52-3** **144138-53-4**
144138-54-5 144138-55-6 **144138-56-7** 144138-57-8
144138-58-9 **144138-59-0** **144138-60-3**
144161-26-2 144161-27-3 144161-28-4
RL: BIOL (Biological study)
(HLA-DR1 antigen-binding, of phage M13 display library)

L34 ANSWER 34 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:566427 HCAPLUS
DOCUMENT NUMBER: 117:166427
TITLE: Specificity determinants of acylaminoacyl-**peptide** hydrolase
AUTHOR(S): Krishna, Radha G.; Wold, Finn
CORPORATE SOURCE: Med. Sch., Univ. Texas, Houston, TX, 77225, USA
SOURCE: Protein Sci. (1992), 1(5), 582-9
CODEN: PRCIEI

DOCUMENT TYPE: Journal
LANGUAGE: English

AB In an attempt to explore how specific features of the substrate's primary structure may affect the activity or rabbit muscle acylaminoacyl-peptide hydrolase (EC 3.4.19.1), a no. of acetylated peptides contg. specific amino acid replacements in specific positions were prepd. and compared as substrates for the hydrolase. The principal variants were D-Ala, Pro, and pos. charges (His, Arg, Lys); in addn., the effect of the length of the peptide was also investigated in a less systematic manner. The substrates were either prepd. by direct acetylation of peptides, by extension of the N-terminus with acetylamino acids or acetylpeptides, activated as N-hydroxysuccinimide esters, or by isolation of the N-terminal peptides from naturally occurring acetylated proteins. It was found that D-Ala on either side of the bond to be cleaved (positions 1 and 2) completely inhibited the enzymic activity, whereas acetylated peptides with D-Ala in positions 3 or 4 were as good substrates as those contg. L-Ala. Peptides with Pro in positions 2 were also inactive, and most of the peptides with Pro in the third position were very poor substrates; only the peptide Ac-AAP (Ac=acetyl, A=amino acid, P=proline) gave reasonably high activity (30% of Ac-AAA), which was reduced to 1-2% if addnl. residues were present at the C-terminus (Ac-AAPA, Ac-AAPAA). The presence of a pos. charge in positions 2, 3, 4, 5, and 6 gave a strong redn. in hydrolase activity varying with the charge's distance from the N-terminus from 0 to 15-20% of the rates obtained with the ref. peptides without pos. charges. Deprotonation of His at high pH generated excellent substrates, and removal of the pos. charges of Lys by acetylation or, even better, succinylation also gave improved substrate quality, demonstrating that the pos. charges are responsible for the inhibition. Long peptides (10-29 residues) were generally found to be poor substrates, esp. when they contained pos. charges and Pro. The better long peptide substrates do not have these residues, but contain neg. charges instead. A survey of the N-terminal **sequences** of more than 100 acetylated proteins revealed that about 95% of them have Pro and/or pos. **charged amino acids** among the first 10 residues, suggesting that these residues may be natural inhibitors of hydrolase action in vivo. In addn. to the specific and large effect of the residues described above on substrate quality, it also appears that there is a general effect of the overall **sequence** of each peptide, and that the specific effects of individual residues are modulated significantly by the environment (context) in which they are expressed.

CC 7-3 (Enzymes)

ST acylaminoacylpeptidase substrate structure specificity; acylaminoacyl **peptide** hydrolase substrate structure specificity

IT Molecular structure-biological activity relationship
(acylaminoacylpeptidase substrate, of **peptides**)

IT **15483-58-6** 41535-86-8 52773-70-3 **52773-71-4**
63769-88-0 72252-79-0 **83201-19-8 119387-21-2**
143786-07-6 **143786-08-7 143786-09-8**
143786-10-1 143786-11-2 **143786-12-3** 143786-13-4
143786-14-5 143786-15-6 143786-16-7
143786-17-8 143786-18-9 143786-19-0
143786-20-3 143786-21-4 143797-53-9
143797-54-0 143797-55-1 143797-56-2 143797-57-3 143797-58-4
RL: BIOL (Biological study)
(acylaminoacylpeptidase specificity for, structure in relation to)

IT **143786-04-3P 143786-05-4P** 143786-06-5P 143797-46-0P
143797-47-1P 143797-48-2P 143797-49-3P
143797-50-6P 143797-51-7P 143797-52-8P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and acylaminoacylpeptidase specificity for, structure in

relation to)

L34 ANSWER 35 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:34563 HCAPLUS
DOCUMENT NUMBER: 116:34563
TITLE: Synthetic **peptides** as modulators of functional responses of intact cells, and their modulation of cytotoxic T-cells
INVENTOR(S): Sitkovsky, Michail V.
PATENT ASSIGNEE(S): United States Dept. of Commerce, USA
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9109613	A1	19910711	WO 1990-US7312	19901219
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
AU 9171423	A1	19910724	AU 1991-71423	19901219
PRIORITY APPLN. INFO.:			US 1989-454827	19891222
			WO 1990-US7312	19901219

AB Methods are provided for selectively inhibiting intracellular biochem. processes leading to cellular responses. The invention provides specifically designed peptides as, e.g., inhibitors or (pseudo)substrates of protein kinases; these peptides are able to strongly inhibit the activation of impermeabilized cells. At least the 1st 2 amino-terminal amino acids of the peptides are pos. charged (lysine or arginine). Thus peptide PKG-S (I), having the sequence RKRSRAE and a peptide substrate of cyclic GMP-dependent protein kinase, strongly inhibited cytotoxic T-lymphocyte (CTL) proliferation. I (.ltoreq.250 .mu.M) had little effect on T-cell receptor-triggered exocytosis, but the same concn. of I inhibited 85% of proliferative response to the same activating ligand, indicating selectivity of response in a concn.-dependent manner. Activities of other peptides of the invention against CTL are included.

IC ICM A61K037-02
ICS C07K005-00; C12N009-00
CC 1-7 (Pharmacology)
Section cross-reference(s): 6, 7
ST kinase pseudosubstrate **peptide** cell modulation; cytotoxic T cell modulation **peptide**; immunomodulator **peptide** kinase inhibitor
IT **Peptides**, biological studies
RL: BIOL (Biological study)
(for cell response inhibition, protein phosphorylation in relation to)
IT Proteins, biological studies
RL: BIOL (Biological study)
(inhibition of, in cell response inhibition, **peptides** for, protein phosphorylation in relation to)
IT Molecular structure-biological activity relationship
(of **peptide** (pseudo)substrates/inhibitors of kinases, on cytotoxic T-cell proliferation)
IT Immunomodulators
(**peptide** (pseudo)substrates/inhibitors of protein kinase as)
IT Cell
(**peptides** for inhibition of response of, protein phosphorylation in relation to)

IT **Amino acids**, biological studies
RL: BIOL (Biological study)
(pos.-**charged**, amino-terminal, in **peptide** for inhibition of intracellular response)
IT Phosphorylation, biological
(protein, **peptides** mimicking phosphorylation sites of, for inhibition of cell response)
IT Lymphocyte
(T-cell, cytotoxic, **peptide** (pseudo)substrate/inhibitor of kinase effect on proliferation and function of)
IT Receptors
RL: BIOL (Biological study)
(TCR (T-cell antigen receptor), interleukin-2 and monoclonal antibody to, cytotoxic T-cell proliferation triggered by, **peptide** (pseudo)substrate/inhibitor of kinase in inhibition of)
IT Antigens
RL: BIOL (Biological study)
(TCR receptors, interleukin-2 and monoclonal antibody to, cytotoxic T-cell proliferation triggered by, **peptide** (pseudo)substrate/inhibitor of kinase in inhibition of)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 2, anti-T-cell receptor monoclonal antibody and, cytotoxic T-cell proliferation triggered by, inhibition of, **peptide** (pseudo)substrate/inhibitor of kinase in)
IT Antibodies
RL: BIOL (Biological study)
(monoclonal, to TCR receptor, interleukin-2 and, cytotoxic T-cell proliferation triggered by, **peptide** (pseudo)substrate/inhibitor of kinase in inhibition of)
IT 56-45-1, Serine, biological studies 60-18-4, Tyrosine, biological studies 72-19-5, Threonine, biological studies
RL: BIOL (Biological study)
(amino acid sequences near, of phosphorylatable protein, **peptides** with, for inhibition of cell response)
IT 56-87-1, L-Lysine, biological studies 74-79-3, L-Arginine, biological studies
RL: BIOL (Biological study)
(amino-terminal, in **peptide** for inhibition of intracellular response)
IT 59587-18-7 **65189-71-1** 81156-93-6 81187-14-6 82801-73-8 **84745-13-1** 87621-34-9 99278-08-7
RL: BIOL (Biological study)
(cytotoxic T-lymphocyte proliferation in presence of)

L34 ANSWER 36 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:675263 HCAPLUS
DOCUMENT NUMBER: 115:275263
TITLE: Isothiocyanic acid mixed anhydride for amino acid thiohydantoin formation and **peptide** sequencing
INVENTOR(S): Hawke, David H.; Boyd, Victoria
PATENT ASSIGNEE(S): Applied Biosystems, Inc., USA
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9109868	A1	19910711	WO 1990-US7567	19901220
W: JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
US 5049507	A	19910917	US 1989-454666	19891221
US 5041388	A	19910820	US 1990-547088	19900629
EP 506846	A1	19921007	EP 1991-902443	19901220
EP 506846	B1	19950719		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 05500421	T2	19930128	JP 1991-502895	19901220

PRIORITY APPLN. INFO.: US 1989-454666 19891221
US 1990-547088 19900629
WO 1990-US7567 19901220

OTHER SOURCE(S): MARPAT 115:275263

AB An N-protected free amino acid or terminal amino acid of a peptide is reacted with a mixed anhydride of isothiocyanic acid and a carboxylic, carbonic, or sulfonic acid, under basic conditions, to produce an amino acid or peptidyl thiohydantoin. The mixed anhydride may be a soln.-phase reagent or a solid-phase reagent. The method may be used for C-terminal peptide sequencing. A solid-phase reagent for use in the method is described. C-terminal anal. of, e.g., leucine-enkephalin (YGGFL) using benzoyl isothiocyanate is described, as is prepn. of an activated solid support.

IC ICM C07K001-10
ICS G01N033-68

CC 9-14 (Biochemical Methods)
Section cross-reference(s): 34

ST amino acid thiohydantoin prepn; isothiocyanate mixed anhydride amino acid thiohydantoin; **peptide** sequencing thiohydantoin

IT Sulfonic acids, compounds
RL: ANST (Analytical study)
(compds. with mixed anhydrides with isothiocyanic acid, for amino acid thiohydantoin formation, **peptide** sequencing in relation to)

IT Ion exchangers
(in **peptide** sequencing, immobilized isothiocyanic acid mixed anhydride for thiohydantoin formation in relation to)

IT Anhydrides
RL: ANST (Analytical study)
(mixed, with isothiocyanic acid, for amino acid thiohydantoin formation, **peptide** sequencing in relation to)

IT Immobilization, biochemical
(of isothiocyanic acid mixed anhydride, for amino acid thiohydantoin formation, **peptide** sequencing in relation to)

IT **Peptides**, analysis
RL: ANST (Analytical study)
(sequencing of carboxyl-terminal amino acids of, isothiocyanic acid mixed anhydride for thiohydantoin formation in)

IT Amino acids, preparation
RL: SPN (Synthetic preparation); PREP (Preparation)
(thiohydantoins, prepn. of, with isothiocyanic acid mixed anhydride, **peptide** sequencing in relation to)

IT 532-55-8, Benzoyl isothiocyanate 1424-53-9, Benzenesulfonyl isothiocyanate 6374-24-9 13250-46-9, Acetyl isothiocyanate 16182-04-0
RL: PRP (Properties)
(at. **charge** of, **amino acid** thiohydantoin formation in relation to)

IT 13588-95-9 14486-05-6, Alanyl methionine **58822-25-6**
RL: ANST (Analytical study)

(carboxyl-terminal sequencing of, benzoyl isothiocyanate in)
IT 463-79-6D, Carbonic acid, mixed anhydrides with isothiocyanic acid 532-55-8, Benzoyl isothiocyanate 3129-90-6D, Isothiocyanic acid, mixed anhydrides with carboxylic or carbonic or sulfonic acids 3129-90-6D, Isothiocyanic acid, trialkylsilyl esters, reaction products
RL: ANST (Analytical study)
(for amino acid thiohydantoin formation, **peptide** sequencing in relation to)
IT 93700-38-0
RL: ANST (Analytical study)
(in carboxyl-terminal **peptide** sequencing)
IT 5765-48-0
RL: ANST (Analytical study)
(in isothiocyanic acid mixed anhydride immobilization on solid support, **peptide** sequencing in relation to)
IT 2290-65-5D, reaction products with activated solid supports 137388-41-1D, reaction products with activated solid supports
RL: ANST (Analytical study)
(in isothiocyanic acid mixed anhydride immobilization, **peptide** sequencing in relation to)

L34 ANSWER 37 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:445093 HCAPLUS
DOCUMENT NUMBER: 115:45093
TITLE: Rational scanning mutagenesis of a protein kinase identifies functional regions involved in catalysis and substrate interactions
AUTHOR(S): Gibbs, Craig S.; Zoller, Mark J.
CORPORATE SOURCE: Dep. Protein Eng., Genentech Inc., South San Francisco, CA, 94080, USA
SOURCE: J. Biol. Chem. (1991), 266(14), 8923-31
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A systematic mutagenesis strategy was used to identify the functional regions and residues of a protein kinase. Clusters of the **charged amino acids** in the catalytic subunit of Saccharomyces cerevisiae cAMP-dependent protein kinase, were systematically mutated to alanine, producing a set of mutations that encompassed the entire mol. Residues indispensable for enzyme activity were identified by testing the ability of the mutants to function in vivo. Active mutants were assayed in vitro, and mutants with reduced specific activity were subsequently analyzed by steady-state kinetics to det. the effects of the mutation on kcat and Km for MgATP and for a peptide substrate. Specific residues and regions of the enzyme were identified that are likely to be important in catalysis and in binding of MgATP, functions that are common to all protein kinases. Addnl. regions were identified that are likely to be important in binding a peptide substrate, the recognition of which is likely to be specific to the serine/threonine protein kinases that have a requirement for basic residues around the target hydroxyamino acid. The properties of mutants defective in substrate recognition were consistent with an ordered sequential reaction mechanism. This represents the first comprehensive anal. of a protein kinase by a rational mutagenesis strategy.
CC 7-5 (Enzymes)
IT **Amino acids**, biological studies
RL: BIOL (Biological study)
(**charged**, of protein kinase, of yeast, alanine substitution for, catalytic and substrate interaction functional regions identification in relation to)

IT Free energy
(of binding, in protein kinase-transition state complexes, **charged amino acid**-to-alanine effects on)
IT 1476-84-2, Magnesium ATP **65189-71-1**, Kemptide
RL: RCT (Reactant)
(reaction of, with protein kinase and mutant forms of yeast, kinetics of)

L34 ANSWER 38 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1990:36417 HCAPLUS
DOCUMENT NUMBER: 112:36417
TITLE: A **charge**-transfer effect in solid phase **peptide** synthesis. Unusually high reactivity in **peptide** bond formation between p-nitrobenzophenone oxime resin ester and **amino acid** 4-(methylthio)phenyl ester
AUTHOR(S): Park, Dong Hyun; Jung, Jae Kyu; Lee, Yoon Sik
CORPORATE SOURCE: Coll. Eng., Seoul Natl. Univ., Seoul, 151-742, S. Korea
SOURCE: Bull. Korean Chem. Soc. (1988), 9(6), 394-8
CODEN: BKCSDE; ISSN: 0253-2964
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Unusually high reactivity was found in peptide bond formation between p-nitrobenzophenone oxime resin (I) and amino acid 4-(methylthio)phenyl (MTP) esters. A charge-transfer complex between the two Ph rings of I and the incoming amino acid MTP esters was considered to be responsible to accelerate the aminolysis reaction of the peptide oxime resin ester. Several di-, tri-, and pentapeptide fragments for prepg. enkephalin and glutathione oligomers were successfully prepd. in short times.
CC 34-3 (Amino Acids, Peptides, and Proteins)
ST charge transfer Merrifield synthesis **peptide**; reactivity nitrobenzophenone oxime resin methylthiophenyl ester
IT Reactivity
(in **peptide** bond formation between nitrobenzophenone oxime resin esters and amino acid (methylthio)phenyl esters)
IT Electron exchange
(in solid-phase **peptide** synthesis using nitrobenzophenone oxime resin esters and amino acid (methylthio)phenyl esters)
IT Merrifield synthesis
(of **peptide** via nitrobenzophenone oxime resin esters and **amino acid** (methylthio)phenyl esters, **charge** -transfer effect in)
IT **Peptides**, preparation
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, by solid-phase method using nitrobenzophenone oxime resin esters and **amino acid** (methylthio)phenyl esters, **charge**-transfer effect in)
IT **Amino acids**, esters
RL: RCT (Reactant)
(esters, with 4-(methylthio)phenol, **peptide** coupling of, with nitrobenzophenone oxime resin esters, **charge**-transfer effect in)
IT 13139-15-6
RL: RCT (Reactant)
(**peptide** coupling of)
IT 17659-11-9
RL: RCT (Reactant)
(**peptide** coupling of, with protected dipeptide ester with

resin-bound nitrobenzophenone oxime)
IT 616-34-2, Glycine methyl ester 1738-68-7, Glycine benzyl ester 6456-74-2, Glycine tert-butyl ester 39047-37-5 39229-42-0, Glycine phenyl ester 124550-15-8, Glycine 4-(methylthio)phenyl ester 124550-16-9, .beta.-Alanine 4-(methylthio)phenyl ester 124550-17-0, Leucine 4-(methylthio)phenyl ester 124550-18-1
RL: RCT (Reactant)
(**peptide** coupling of, with protected phenylalanine ester with resin-bound nitrobenzophenone oxime)
IT 17646-23-0
RL: RCT (Reactant)
(**peptide** coupling of, with protected tyrosine ester with resin-bound nitrobenzophenone oxime)
IT 4530-37-4DP, ester with resin-bound nitrobenzophenone oxime 5068-28-0DP, ester with resin-bound nitrobenzophenone oxime
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and **peptide** coupling of)
IT 13734-34-4DP, N-tert-Butoxycarbonyl-L-phenylalanine, ester with resin-bound nitrobenzophenone oxime
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and **peptide** coupling of, with amino acid (methylthio)phenyl esters)
IT 2130-96-3DP, ester with resin-bound nitrobenzophenone oxime 124568-71-4DP, ester with resin-bound nitrobenzophenone oxime
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and **peptide** coupling of, with glycine (methylthio)phenyl ester)
IT **124550-19-2DP**, ester with resin-bound nitrobenzophenone oxime
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and **peptide** coupling of, with lysine (methylthio)phenyl ester)
IT 7625-57-2P 23547-47-9P 42280-29-5P 124447-39-8P 124550-06-7P 124550-07-8P 124550-08-9P 124550-09-0P 124550-10-3P 124550-11-4P **124550-12-5P** 124550-13-6P 124550-14-7P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, using nitrobenzophenone oxime resin ester for **peptide** bond formation)
IT 4530-20-5 5672-81-1
RL: RCT (Reactant)
(solid-phase **peptide** coupling of)

L34 ANSWER 39 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1990:34323 HCAPLUS
DOCUMENT NUMBER: 112:34323
TITLE: **Peptide** determinant associated with immunity and stimulating to T-cells
INVENTOR(S): Steinman, Lawrence; Zamvil, Scott
PATENT ASSIGNEE(S): Leland Stanford Junior University, USA
SOURCE: Eur. Pat. Appl., 7 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 304279	A2	19890222	EP 1988-307608	19880817
EP 304279	A3	19900516		
EP 304279	B1	19971105		

R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE

CA 1340012	A1	19980825	CA 1988-574902	19880816
JP 01131124	A2	19890524	JP 1988-203305	19880817
EP 805162	A1	19971105	EP 1997-106788	19880817

R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE

AT 159952	E	19971115	AT 1988-307608	19880817
ES 2109217	T3	19980116	ES 1988-307608	19880817

PRIORITY APPLN. INFO.: US 1987-86694 19870817
EP 1988-307608 19880817

AB Peptides are provided comprising **sequences** from proteins of interest which include a motif comprising a **charged amino acid**, followed by 2 hydrophobic amino acids, followed in the next or succeeding position by a polar amino acid, where the charged or polar amino acid may be substituted by glycine. Peptides having the motif are stimulatory to T-cells and allow for modulation of immune response, as illustrated by making the host tolerant to neurol. proteins (no data). Peptides P35-47 of the human myelin basic protein (MBP) comprising the Arg-Phe-Phe-Ser motif and P5-17 of human MBP comprising the Lys-Tyr-Leu-Ala-Thr motif stimulated mouse T-cells restricted by major histocompatibility complex I-E.alpha.uE.beta.u and I-A.alpha.sA.beta.u or I-A.alpha.uA.beta.u, resp., in a proliferative response assay. Peptide P89-101 of human MBP comprising the motif His-Phe-Phe-Lys stimulated mouse T-cells restricted by I-A.alpha.sA.beta.s.

IC ICM C07K007-04
ICS A61K037-02

CC 15-10 (Immunochemistry)

ST **peptide** determinant T lymphocyte stimulation; human myelin basic protein **peptide** immunostimulant

IT **Peptides**, biological studies
RL: BIOL (Biological study)
(T-cell stimulation in myasthenics with, of acetylcholine receptor)

IT Myasthenia gravis
(T-cell stimulation in, with synthetic **peptide** of acetylcholine receptor)

IT Lymphocyte
(conjugates with **peptide**, for making human host tolerant to neuropeptide)

IT Antigens
RL: BIOL (Biological study)
(oligopeptide contg. **peptide** restricted by transplantation antigen and epitope of)

IT Immunostimulants
(**peptides** contg. determinant of immunogen restricted by transplantation antigen)

IT Immunomodulators
(polypeptides contg. **sequence** of immunogen restricted by transplantation antigen)

IT Proteins, biological studies
RL: BIOL (Biological study)
(transplantation antigen-restricted **peptide sequence** -contg., as immunomodulator)

IT Phospholipoproteins
RL: BIOL (Biological study)
(MBP (myelin basic protein), **peptide** determinant of, T-cell response to)

IT Lymphocyte
(T-, stimulation of, with **peptides**)

IT **Peptides**, compounds
RL: BIOL (Biological study)

(conjugates, with lymphocyte, for making human host tolerant to neuropeptide)
IT Antigens
RL: BIOL (Biological study)
(major histocompatibility complex, T-cells restricted by, stimulation of, **peptides** for)
IT **Peptides**, biological studies
RL: BIOL (Biological study)
(oligo-, restricted T-cells stimulation with)
IT Pharmaceutical dosage forms
(solns., neurotolerization of human host to, **peptides** for)
IT Antigens
RL: BIOL (Biological study)
(transplantation, immunogen restricted by, oligopeptide contg. **sequence** of, as immunomodulator)
IT **Peptides**, biological studies
RL: BIOL (Biological study)
(N-Ac, of immunogen amino-terminus, immune response of human modulation by)
IT 124470-29-7
RL: BIOL (Biological study)
(T-cell stimulation in myasthenics with, as synthetic **peptide** of acetylcholine receptor)
IT 124470-31-1 124470-32-2 124470-33-3
RL: BIOL (Biological study)
(**peptide** contg., T-cell stimulation with)
IT 124470-28-6 **124470-30-0**
RL: BIOL (Biological study)
(**peptide** determinant on, T-cells stimulation with)
IT 51-84-3, Acetylcholine, biological studies
RL: BIOL (Biological study)
(receptor for, synthetic **peptide** of, T-cell stimulation in myasthenics with)

L34 ANSWER 40 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:627463 HCAPLUS
DOCUMENT NUMBER: 111:227463
TITLE: Circular dichroism studies on synthetic signal **peptides** indicate .beta.-conformation as a common structural feature in highly hydrophobic environment
AUTHOR(S): Reddy, G. Laxma; Nagaraj, R.
CORPORATE SOURCE: Cent. Cell. Mol. Biol., Hyderabad, 500 007, India
SOURCE: J. Biol. Chem. (1989), 264(28), 16591-7
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The conformations of synthetic peptides corresponding to signal **sequences** of chicken lysozyme and Escherichia coli proteins alk. phosphatase and lipoprotein (wild-type) and their variants with a **charged amino acid** in the hydrophobic region, have been studied by CD spectroscopy in trifluoroethanol and micelles of SDS, Brij 35, and Na deoxycholate. In trifluoroethanol and aq. mixts. f trifluoroethanol, the wild-type and variant signal **sequences** show similar conformational behavior. The wild-type signal peptides show increasing amts. of .beta.-structure going from SDS to deoxycholate micelles (i.e. increasing order of hydrophobicity). The variant signal **sequences**, however, are largely unordered in micelles. The absence of .beta.-structure in variant signal **sequence** which do not initiate protein translocation across membranes, strongly suggests

that the ability of signal **sequences** to adopt .beta.-structure in a highly hydrophobic environment is important for function.
CC 6-3 (General Biochemistry)
ST signal **peptide** conformation hydrophobic environment
IT Lipoproteins
RL: BIOL (Biological study)
(signal **peptide** of, conformation of, targeting in relation to)
IT Conformation and Conformers
(secondary, of signal **peptide**, alk. phosphatase and lysozyme and lipoprotein targeting in relation to)
IT **Peptides**, properties
RL: PRP (Properties)
(signal, conformation of, of alk. phosphatase and lysozyme and lipoprotein, targeting in relation to)
IT Biological transport
(translocation, signal **peptide** conformation in alk. phosphatase and lysozyme and lipoprotein in relation to)
IT 2389-45-9 2488-15-5 3262-72-4 5068-28-0 13139-15-6 15761-38-3 15761-39-4
RL: RCT (Reactant)
(**peptide** coupling reaction with **peptide**)
IT 64152-76-7P **123899-18-3P** 123899-23-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and demethylation of)
IT 37783-45-2P 43189-51-1P 53613-22-2P **101708-34-3P** 123899-12-7P **123899-14-9P** 123899-16-1P 123899-21-8P 123899-25-2P 123899-26-3P 123899-28-5P 123899-29-6P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and deprotection of)
IT **123899-19-4P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction with dodecapeptide protected deriv.)
IT **123913-62-2P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction with hexapeptide protected deriv.)
IT **123899-15-0P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction with lysine protected deriv.)
IT 82882-78-8P **123899-32-1P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction with pentapeptide protected deriv.)
IT 112725-58-3P 123899-30-9P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction with **peptide** protected deriv.)
IT 75-89-8 151-21-3, SDS, biological studies 302-95-4, Sodium deoxycholate 9002-92-0, Brij 35
RL: PRP (Properties)
(signal **peptide** conformation in, alk. phosphatase and lysozyme and lipoprotein targeting in relation to)
IT 9001-63-2, Lysozyme 9001-78-9, Alkaline phosphatase
RL: BIOL (Biological study)
(signal **peptide** of, conformation of, targeting in relation to)

L34 ANSWER 41 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:627401 HCAPLUS
DOCUMENT NUMBER: 111:227401
TITLE: On the interactions of charged side chains with the .alpha.-helix backbone

AUTHOR(S): Godzik, Adam; Wesolowski, Tomasz
CORPORATE SOURCE: Inst. Exp. Phys., Univ. Warsaw, Warsaw, 02-089, Pol.
SOURCE: Biophys. Chem. (1988), Volume Date 1987, 31(1-2), 29-34
CODEN: BICIAZ; ISSN: 0301-4622
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of the position of **charged amino acid** side chains on the stability of the .alpha.-helix are investigated. Calcns. for the model poly(alanine) 13 residue .alpha.-helix, with modifications based on exptl. work, are performed at 3 levels of approxn. The obsd. stabilization of the .alpha.-helix could be explained by interactions between its macrodipole and **charged amino acid** side chains. Limitations of the model are discussed.

CC 6-3 (General Biochemistry)

IT Dipole moment
(of poly(alanine) .alpha.-helical conformation, **charged amino acid** residues interaction with, RNase AC-**peptide** in relation to)

IT Free energy
(conformational, of poly(alanine), **charged amino acid** substitution effect on, RNase AC-**peptide** in relation to)

IT Force
(electrostatic, attractive, of poly(alanine) **peptide** backbone with substituted **charged amino acids**, .alpha.-helical conformation stability in relation to)

IT Conformation and Conformers
(.alpha.-helical, of proteins, **charged amino acid** residues interaction with **peptide** backbone in, RNase AC-**peptide** in relation to)

IT 118593-57-0 **118593-58-1 118593-59-2**
118593-60-5 118593-61-6 118593-62-7
118593-63-8 118593-64-9 118593-65-0
118593-66-1 118593-67-2 118593-68-3
118593-69-4 118593-70-7 **118593-71-8 118593-72-9**
118593-73-0 118593-74-1 118593-75-2
118593-76-3 118593-77-4 118593-78-5
118593-79-6 118593-80-9 118593-81-0
118593-82-1 **118597-11-8**
RL: PRP (Properties)
(conformational energy of, calcn. of, RNase AC-**peptide** in relation to)

IT 25191-17-7, Poly(alanine) 25213-34-7, Poly(alanine)
RL: PRP (Properties)
(.alpha.-helical conformation of, **charged amino acid** residue interaction with **peptide** backbone in)

IT 84325-24-6
RL: PRP (Properties)
(.alpha.-helical conformation of, glutamate-2 and histidine-12 stabilization of, energy calcns. for model **peptides** in relation to)

L34 ANSWER 42 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:592279 HCAPLUS
DOCUMENT NUMBER: 111:192279
TITLE: Chargerins from rat liver mitochondria
INVENTOR(S): Higuchi, Tomihiko; Oita, Kenji
PATENT ASSIGNEE(S): Sumitomo Chemical Co., Ltd., Japan

SOURCE: Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 63186000	A2	19880801	JP 1987-16497	19870126

AB Rat liver mitochondria are extd. with CHCl3-MeOH (2:1) to give a hydrophobic protein (chargerin) having a mol. wt. of .apprx.12,000 and a partial amino acid sequence of Ile-Tyr-Leu-Pro-Leu-Ser-Leu-Pro-Pro. Chargerin is essential for energy transduction in oxidative phosphorylation, specifically binds to antilabeled chargerin II antibody, and is useful in biochem. application (e.g. research).
IC ICM C07K015-12
ICA C07K003-12; C12P021-00; C12P021-02
CC 13-1 (Mammalian Biochemistry)
Section cross-reference(s): 9
IT **123498-75-9**
RL: BIOL (Biological study)
(partial **amino acid** sequence of **chargerin** from rat liver mitochondria)

L34 ANSWER 43 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1987:28978 HCAPLUS
DOCUMENT NUMBER: 106:28978
TITLE: Interaction of a synthetic fragment of p21ras with cellular proteins
AUTHOR(S): Chertov, O. Yu.; Khokhlachev, A. V.; Deigin, V. I.
CORPORATE SOURCE: M. M. Shemyakin Inst. Bioorg. Chem., Moscow, USSR
SOURCE: Bioorg. Khim. (1986), 12(9), 1157-63
CODEN: BIKHD7
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB A decapeptide corresponding to residues 35-44 (-Thr-Ile-Glu-Asp-Ser-Tyr-Arg-Lys-Gln-Val-) of p21ras was synthesized. This peptide caused pptn. of some proteins from the Triton X-100 lysate of NIH 3T3 EJ cells. SDS-PAGE demonstrated the presence of many proteins in the ppt. Peptide labeled with 125I and Bolton-Hunter reagent specifically recognized 4 proteins of mol. wt. 27, 35, 50, and 85 kilodaltons (kDa). The order of **charged amino acid** residues in the fragment 35-44 of p21ras (Glu-Asp-X-X-Arg-Lys-X-) is complementary to that of the substrate **sequence** of tyrosine-specific protein kinases (-Arg-X-X-Glu-Asp-X-X-Tyr-) (X is an amino acid). It is suggested that p21ras proteins directly regulate phosphorylation of the target proteins of these kinases. A model for functioning of p21ras proteins predicts the presence in their structure of certain sites homologous to **sequences** recognizable by tyrosine-specific kinases. Indeed, 2 such sites are present in the **sequence** of all p21ras proteins, namely the residues 88-92 and 104-108.
CC 6-3 (General Biochemistry)
Section cross-reference(s): 7
IT Fibroblast
(proteins of, **peptide** of protein p21ras binding of)
IT **106009-37-4P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and C terminus deprotection of)
IT **106001-23-4P**

RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and N-terminus deprotection of)
IT **106019-58-3P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and deprotection)
IT **106009-31-8P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. and fibroblast proteins binding of)
IT 3392-08-3P 15387-45-8P 21760-98-5P 50903-59-8P 55878-15-4P
57866-90-7P 70989-89-8P 82825-48-7P 87379-92-8P 106001-18-7P
106001-24-5P 106009-38-5P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and **peptide** coupling reaction of)
IT 87218-87-9P 87218-89-1P 106001-19-8P **106001-20-1P**
106001-21-2P 106001-22-3P 106009-32-9P 106009-33-0P 106009-34-1P
106009-35-2P **106009-36-3P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction with protected amino acid deriv.)
IT 80449-02-1, Tyrosine-specific protein kinase
RL: BIOL (Biological study)
(protein p21ras **sequence** homol. with substrates of)

L34 ANSWER 44 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:451954 HCAPLUS
DOCUMENT NUMBER: 103:51954
TITLE: Amino acid **sequence** specificities of an adhesive recognition signal
AUTHOR(S): Yamada, Kenneth M.; Kennedy, Dorothy W.
CORPORATE SOURCE: Lab. Mol. Biol., Natl. Cancer Inst., Bethesda, MD, 20205, USA
SOURCE: J. Cell. Biochem. (1985), 28(2), 99-104
CODEN: JCEBD5; ISSN: 0730-2312
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Synthetic peptides derived from the cell-binding domain of fibronectin have previously been found to inhibit fibronectin-mediated adhesion in vitro competitively and reversibly, as well as inhibiting cell migratory events in vivo. The amino acid **sequence** specificity required for this inhibitory activity was examd. further by using variations of the originally identified active peptide **sequences**. The most active small peptide was the pentapeptide Gly-Arg-Gly-Asp-Ser; although the tetrapeptide Arg-Gly-Asp-Ser retained substantial activity, it was .apprx.3-fold less active. An inverted peptide **sequence** with these same 4 amino acids arranged in the mirror sym. **sequence** Ser-Asp-Gly-Arg was nearly as active as the forward **sequence**. However, the same inverted tetrapeptide **sequence** embedded in a synthetic decapeptide derived from a **sequence** of histocompatibility antigens had minimal activity, suggesting the importance of adjacent **sequences** in modifying the activity of such peptides. Neither substitution of amino acids of the same charge nor reversal of the positions of the 2 **charged amino acids** retains biol. activity. Decreasing the spacing between the charged residues also caused a loss of activity. The adhesive recognition signal thus appears to consist of a specific arrangement of 1 acidic and 1 basic charged group and addnl. information provided by adjacent amino acids.
CC 13-6 (Mammalian Biochemistry)
Section cross-reference(s): 6
ST adhesion recognition **peptide sequence**
IT **Peptides**, biological studies

RL: BIOL (Biological study)
(cell adhesion recognition signal activity of)
IT Fibronectins
RL: BIOL (Biological study)
(cell adhesion recognition signal of, **sequence** specificity of)
IT Protein **sequences**
(of cell adhesion recognition signals, specificity in)
IT Animal cell
(BHK, adhesion of, protein **sequence** of recognition signals for)
IT Adhesion
(bio-, amino acid **sequence** specificity of recognition signal for)
IT Molecular structure-biological activity relationship
(cell adhesion-promoting, of fibronectin-related **peptides**)
IT 91037-65-9 91575-25-6 **96426-21-0** 97461-81-9 97461-82-0 97461-83-1 **97461-84-2** **97461-85-3** 97461-86-4
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(cell adhesion recognition signal activity of)

L34 ANSWER 45 OF 45 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:405244 HCAPLUS
DOCUMENT NUMBER: 101:5244
TITLE: Essential structural requirements for triggering of mast cells by a synthetic **peptide** comprising a **sequence** in the C.epsilon.4 domain of human IgE
AUTHOR(S): Stanworth, D. R.; Coleman, J. W.; Khan, Zahida
CORPORATE SOURCE: Med. Sch., Univ. Birmingham, Birmingham, B15 2TJ, UK
SOURCE: Mol. Immunol. (1984), 21(3), 243-7
CODEN: MOIMD5; ISSN: 0161-5890
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A range of synthetic analogs of the peptides Lys-Thr-Lys-Gly-Ser-Gly-Phe-Phe-Val-PheNH2 (human IgE .epsilon.-chain 497-506 decapeptide) and Lys-Thr-Lys-Gly-Ser-Gly-Phe-PheNH2 (.epsilon.-chain 497-504 octapeptide) were tested for activity as releasers of 5-hydroxytryptamine from rat peritoneal mast cells. The following structural modifications were found to abrogate activity: N-acetylation of the .alpha.-amino group of the N-terminal lysine residue; substitution of the 2 lysine residues by either serine or glutamine; depletion of the 2 C-terminal hydrophobic residues (Val-Phe) of the decapeptide; and substitution of phenylalanine by alanine in the C-terminal position of the octapeptide. These observations point to a requirement for pos. **charged amino acids** and hydrophobic amino acids at the N- and C-terminus, resp., for triggering of mast cells by these short-chain peptides. Releasing activity also depended on the stereospecific conformation of the pos. charged region, since substitution of L-isomeric amino acids by D-isomeric forms of the 3 N-terminal positions of the decapeptide led to loss of potency. Inactive analogs of the decapeptide and octapeptide, at concns. up to 10-4M, failed to antagonize the mediator-releasing effects of the active decapeptide at concns. of 3 .times. 10-6-10-4M.
CC 15-3 (Immunochemistry)
ST IgE **peptide** mast cell triggering
IT **Peptides**, biological studies
RL: BIOL (Biological study)
(of IgE const. domain, of human, structural requirements for mast cell triggering by)

IT Mast cell
(triggering of, by IgE of human, **peptide** structure of const. domain requirements for)
IT Immunoglobulins
RL: BIOL (Biological study)
(E, **peptides** of const. region of, structural requirements for mast cell triggering by, of human)
IT Molecular structure-biological activity relationship
(serotonin release-stimulating, of IgE const. domain **peptides** of human)
IT 90274-60-5 90274-61-6 90274-62-7 90274-63-8 90274-64-9
90274-65-0 90274-66-1 90274-67-2 90274-68-3 90274-69-4
90274-70-7 90274-71-8 90274-72-9 90274-73-0 **90274-74-1**
90364-66-2
RL: BIOL (Biological study)
(of IgE const. domain of human, mast cell triggering by, structure requirements for)

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E56 THROUGH E95 ASSIGNED

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FILE 'REGISTRY' ENTERED AT 09:51:01 ON 10 JUN 2002
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STRUCTURE FILE UPDATES: 7 JUN 2002 HIGHEST RN 427375-75-5
DICTIONARY FILE UPDATES: 7 JUN 2002 HIGHEST RN 427375-75-5

TSCA INFORMATION NOW CURRENT THROUGH January 7, 2002

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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L37  ANSWER 1 OF 40  REGISTRY  COPYRIGHT 2002 ACS
RN   256480-61-2  REGISTRY
CN   L-Alanine, L-alanyl-L-.alpha.-glutamyl-L-lysyl-L-phenylalanyl- (9CI)  (CA
     INDEX NAME)
OTHER NAMES:
CN   6: PN: WO0005250 SEQID: 6 claimed sequence
FS   PROTEIN SEQUENCE; STEREOSEARCH
SQL  5

SEQ3      1 Ala-Glu-Lys-Phe-Ala
            ===             ===
HITS AT:    1, 5

MF   C26 H40 N6 O8
SR   CA
LC   STN Files:   CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.
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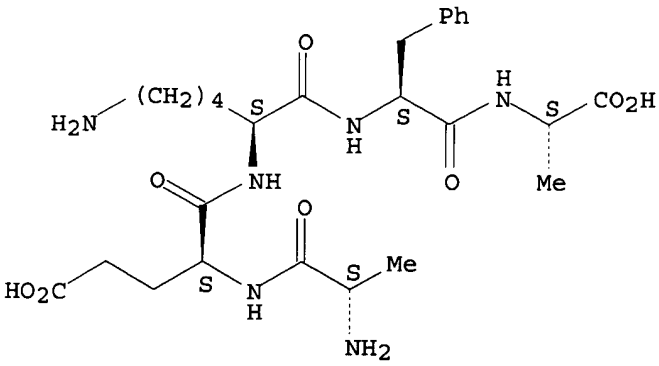


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2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 2 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **256480-60-1** REGISTRY
CN L-Valine, L-alanyl-L-.alpha.-glutamyl-L-lysyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **4**

SEQ3 1 Ala-Glu-Lys-Val
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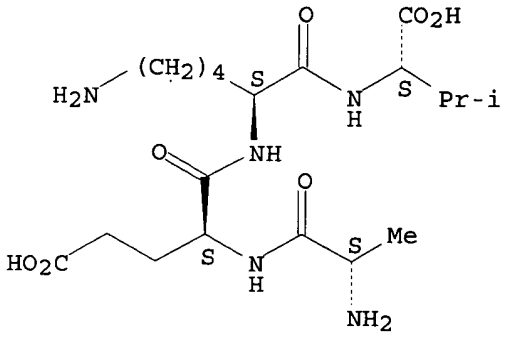
HITS AT: 1, 4

MF C19 H35 N5 O7
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER

Absolute stereochemistry.



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1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 3 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **256480-59-8** REGISTRY
CN L-Alanine, L-alanyl-L-.alpha.-glutamyl-L-lysyl-L-valyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5: PN: WO0005250 SEQID: 5 claimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **5**

SEQ3 1 Ala-Glu-Lys-Val-Ala
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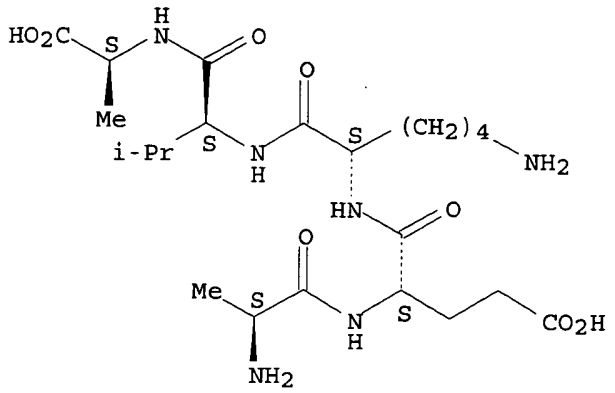
HITS AT: 1, 5

MF C22 H40 N6 O8
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



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L37 ANSWER 4 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **256480-58-7** REGISTRY
CN L-Alanine, L-alanyl-L-.alpha.-glutamyl-L-lysyl-L-tyrosyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 4: PN: WO0005250 SEQID: 4 claimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **5**

SEQ3 1 Ala-Glu-Lys-Tyr-Ala
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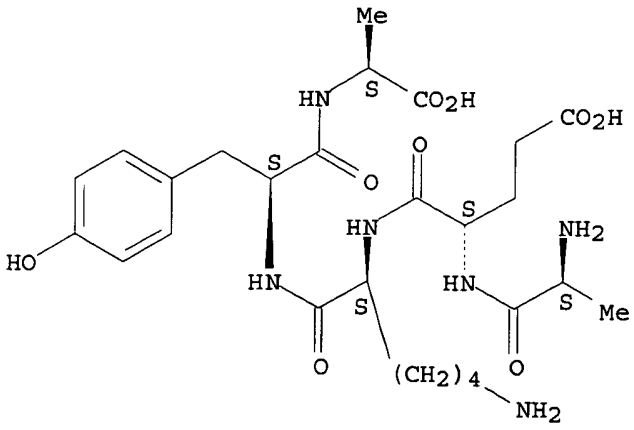
HITS AT: 1, 5

MF C26 H40 N6 O9
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



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L37 ANSWER 5 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-60-1** REGISTRY
CN L-Threonine, L-seryl-L-seryl-L-.alpha.-aspartyl-L-valyl-L-prolyl-L-cysteinyl-L-.alpha.-aspartyl-L-alanyl-L-threonyl-L-leucyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **11**

SEQ3 1 Ser-Ser-Asp-Val-Pro-Cys-Asp-Ala-Thr-Leu-
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11 Thr
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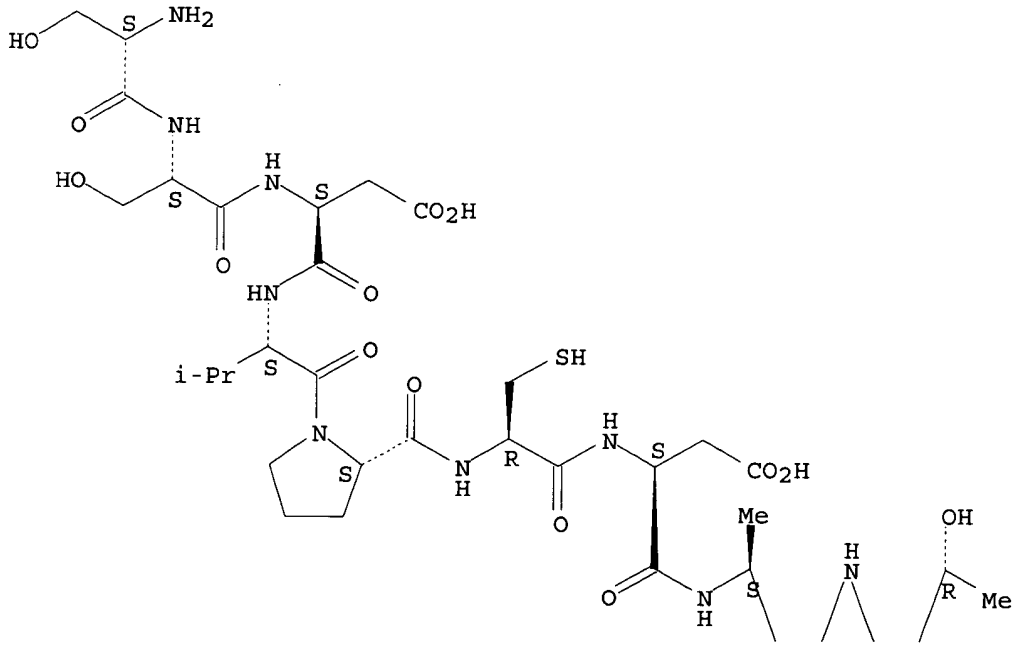
HITS AT: 1, 11

MF C44 H73 N11 O20 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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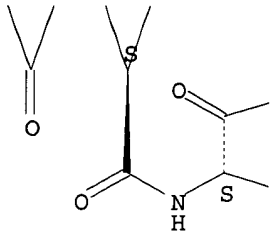

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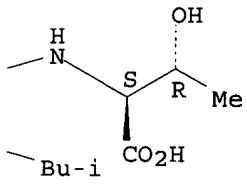

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 6 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-59-8** REGISTRY
CN L-Serine, L-tyrosylglycyl-L-arginyl-L-alanyl-L-.alpha.-aspartyl-L-cysteinyl-L-isoleucyl-L-threonyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Tyr-Gly-Arg-Ala-Asp-Cys-Ile-Thr-Ser
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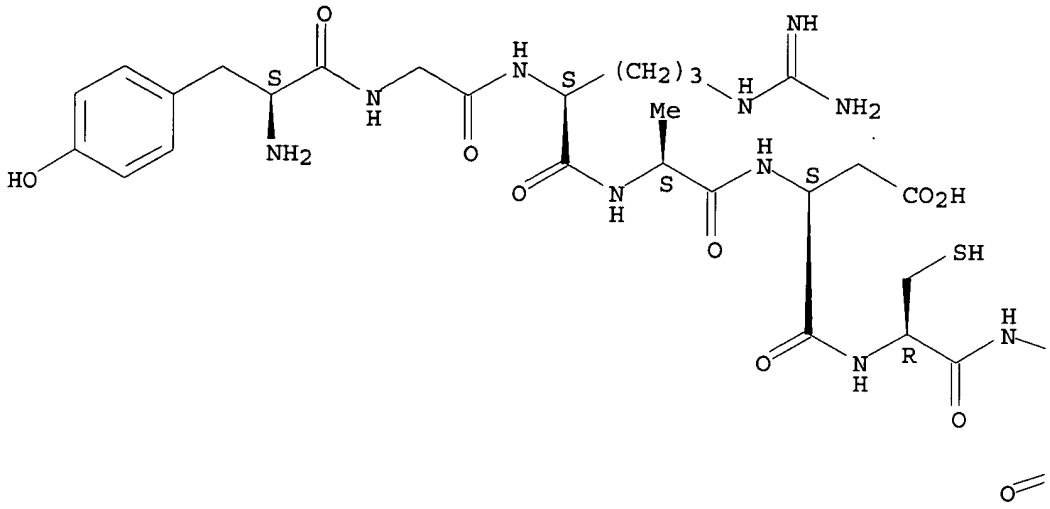
HITS AT: 1, 9

MF C40 H64 N12 O15 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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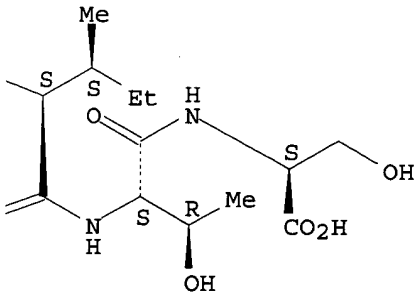

1 REFERENCES IN FILE CA (1967 TO DATE)
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L37 ANSWER 7 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-58-7** REGISTRY
CN L-Serine, L-tryptophylglycyl-L-arginyl-L-alanyl-L-.alpha.-aspartyl-L-cysteinylglycyl-L-isoleucyl-L-threonyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **10**

SEQ3 1 Trp-Gly-Arg-Ala-Asp-Cys-Gly-Ile-Thr-Ser
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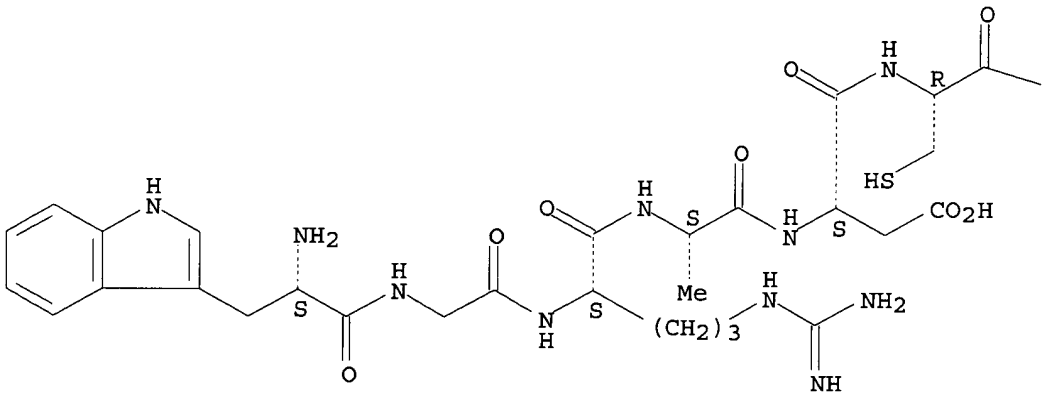
HITS AT: 1, 10

MF C44 H68 N14 O15 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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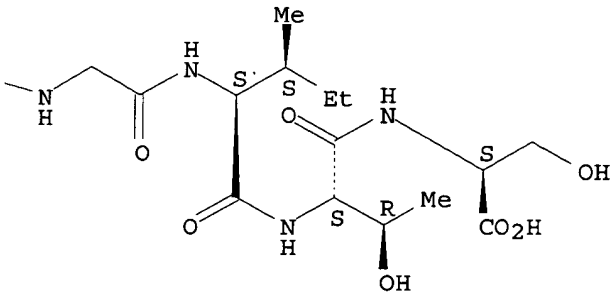

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 8 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-56-5** REGISTRY
CN L-Serine, L-tyrosylglycyl-L-arginyl-L-alanyl-L-.alpha.-aspartyl-L-cysteinylglycyl-L-isoleucyl-L-threonyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **10**

SEQ3 1 Tyr-Gly-Arg-Ala-Asp-Cys-Gly-Ile-Thr-Ser
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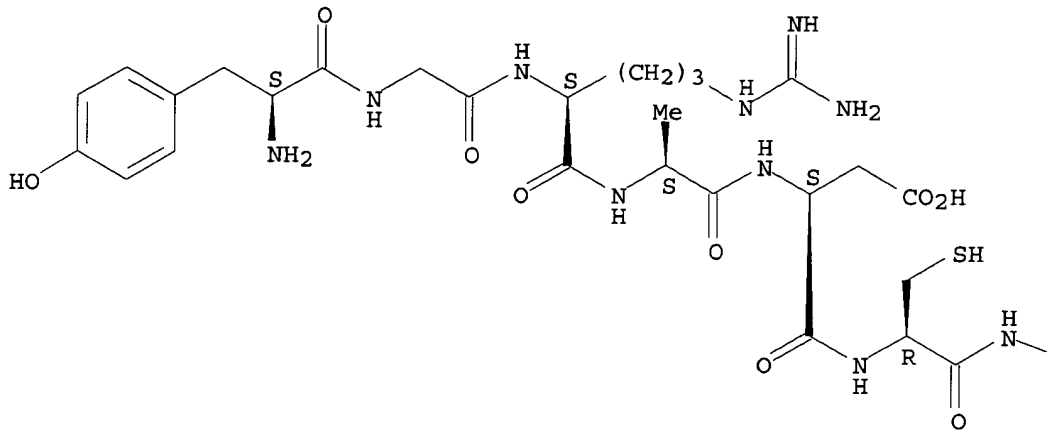
HITS AT: 1, 10

MF C42 H67 N13 O16 S

SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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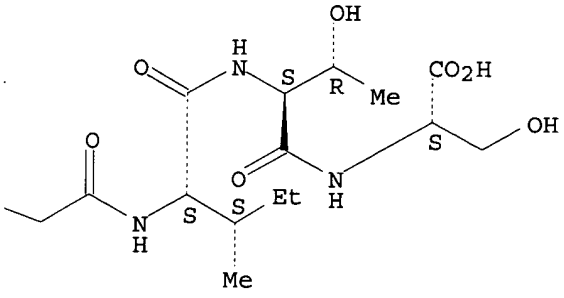

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1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 9 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-54-3** REGISTRY
CN L-Threonine, L-alanylglycyl-L-phenylalanyl-L-asparaginyl-L-leucyl-L-leucyl-L-methionyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **8**

SEQ3 1 Ala-Gly-Phe-Asn-Leu-Leu-Met-Thr
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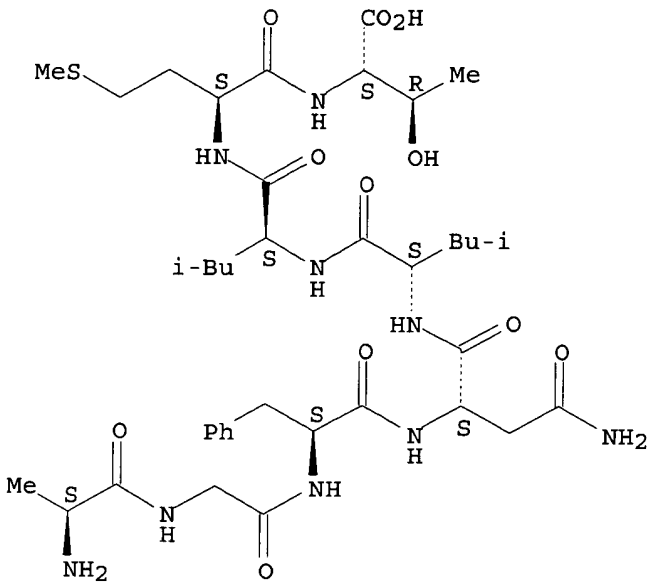
MF C39 H63 N9 O11 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.



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1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 10 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-52-1** REGISTRY
CN L-Threonine, L-isoleucyl-L-valyl-L-isoleucyl-L-valyl-L-.alpha.-aspartyl-L-isoleucyl-L-cysteinyl-L-isoleucyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ile-Val-Ile-Val-Asp-Ile-Cys-Ile-Thr
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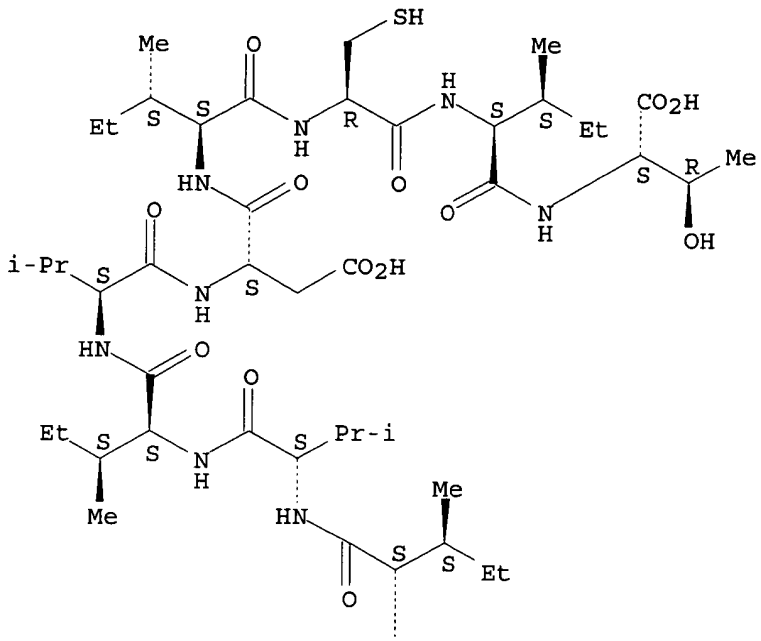
MF C45 H81 N9 O13 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

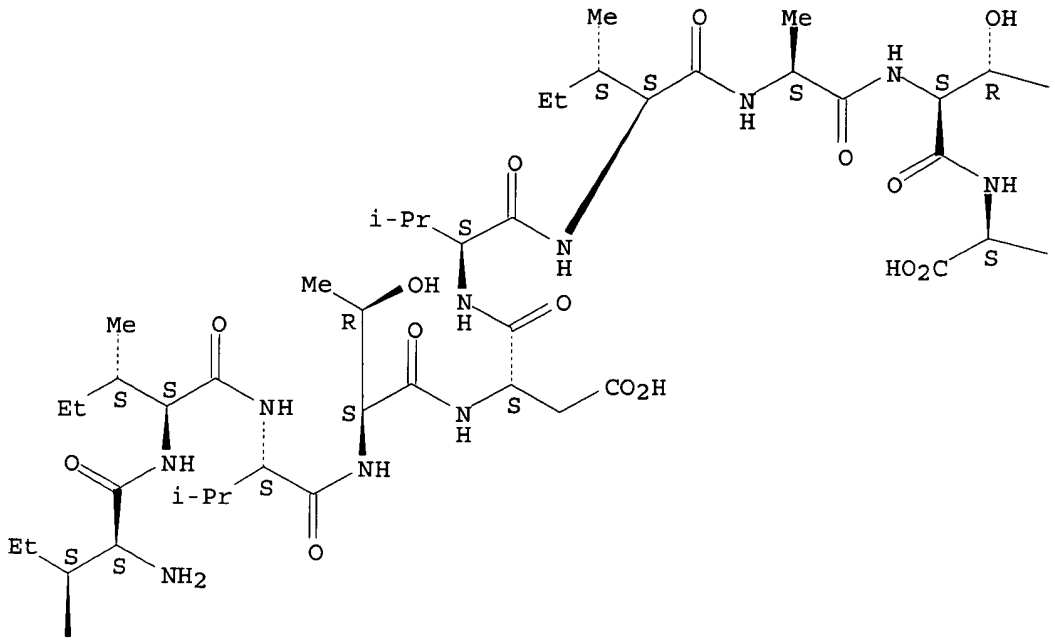
L37 ANSWER 11 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-51-0** REGISTRY
CN L-Leucine, L-isoleucyl-L-isoleucyl-L-valyl-L-threonyl-L-.alpha.-aspartyl-L-valyl-L-isoleucyl-L-alanyl-L-threonyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **10**

SEQ3 1 Ile-Ile-Val-Thr-Asp-Val-Ile-Ala-Thr-Leu
=== ===
HITS AT: 1, 10

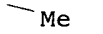
MF C49 H88 N10 O15
SR CA
LC STN Files: CA, CAPLUS

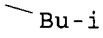
Absolute stereochemistry.

PAGE 1-A



PAGE 1-B





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1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

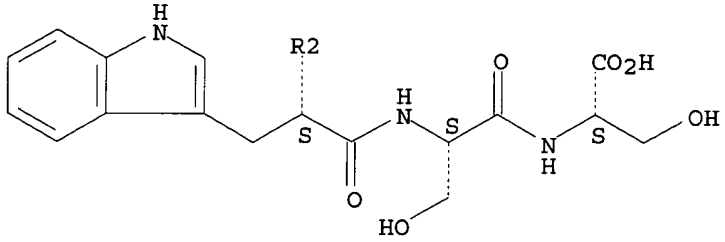
L37 ANSWER 12 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-50-9** REGISTRY
CN L-Serine, L-leucyl-L-leucyl-L-methionyl-L-threonyl-L-leucyl-L-arginyl-L-leucyl-L-tryptophyl-L-seryl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **10**

SEQ3 1 Leu-Leu-Met-Thr-Leu-Arg-Leu-Trp-Ser-Ser
=== ===
HITS AT: 1, 10

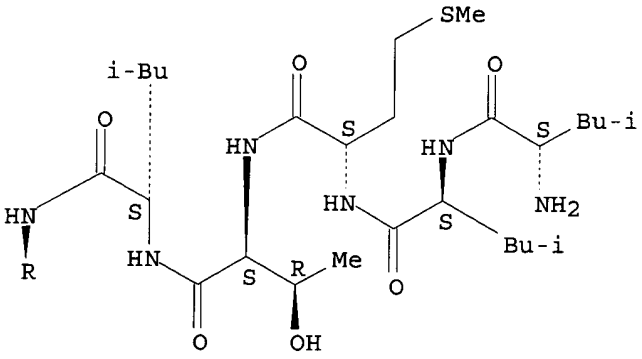
MF C56 H94 N14 O14 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 2-A



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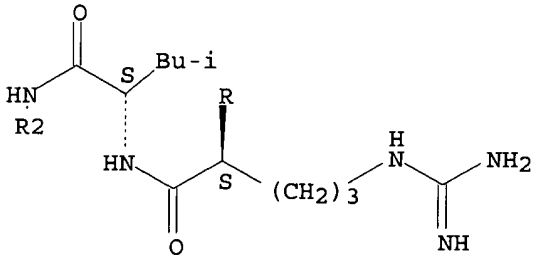


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 13 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **200556-46-3** REGISTRY

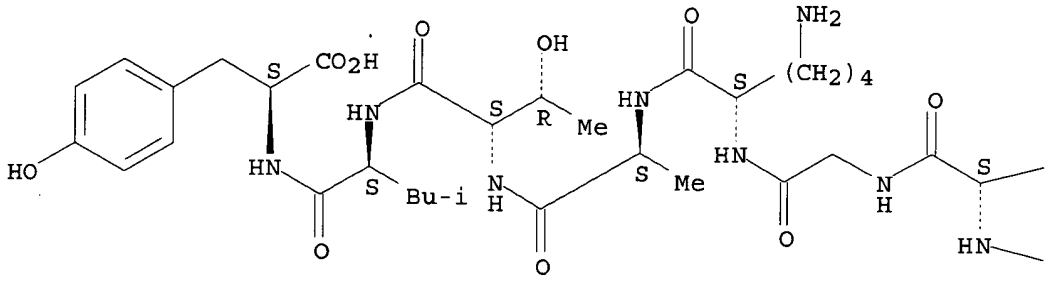
CN L-Tyrosine, L-isoleucyl-L-leucyl-L-leucylglycyl-L-lysyl-L-alanyl-L-threonyl-L-leucyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ile-Leu-Leu-Gly-Lys-Ala-Thr-Leu-Tyr
=== ===
HITS AT: 1, 9

MF C48 H82 N10 O12
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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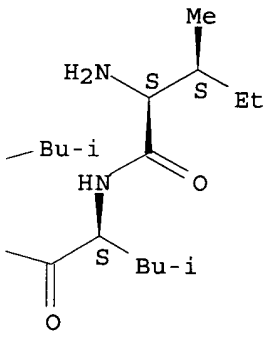


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 14 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **186487-70-7** REGISTRY
CN L-Valine, L-leucylglycyl-L-isoleucyl-L-leucyl-L-leucyl-L-leucyl-L-lysyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **8**

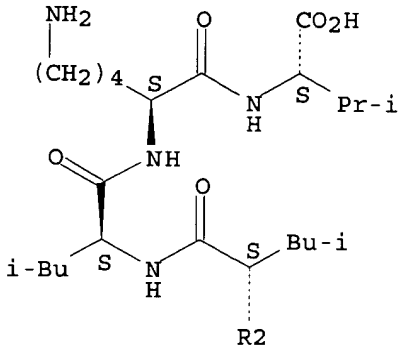
SEQ3 1 Leu-Gly-Ile-Leu-Leu-Leu-Lys-Val
=== ===

HITS AT: 1, 8

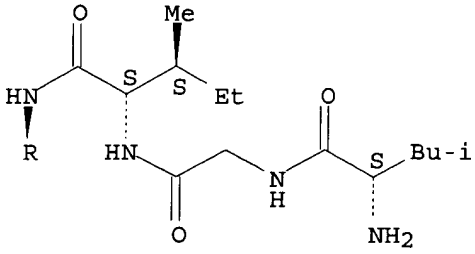
MF C43 H81 N9 O9
SR CA
LC STN Files: CA, CAPLUS

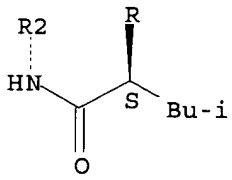
Absolute stereochemistry.

PAGE 1-A



PAGE 2-A





2 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

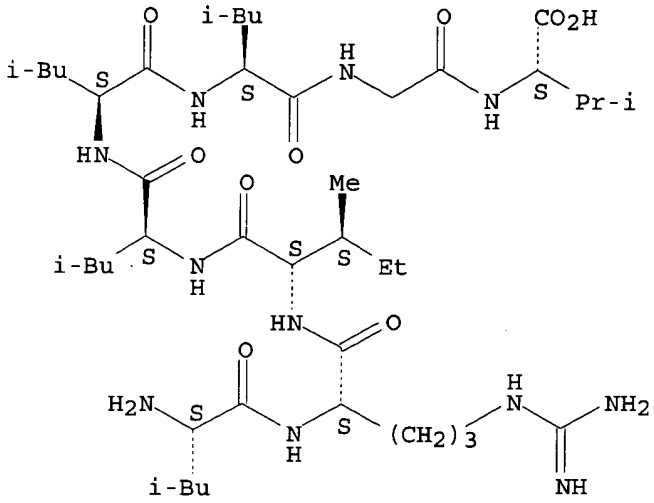
L37 ANSWER 15 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **186487-64-9** REGISTRY
CN L-Valine, L-leucyl-L-arginyl-L-isoleucyl-L-leucyl-L-leucyl-L-leucylglycyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **8**

SEQ3 1 Leu-Arg-Ile-Leu-Leu-Leu-Gly-Val
=== ===

HITS AT: 1, 8

MF C43 H81 N11 O9
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.



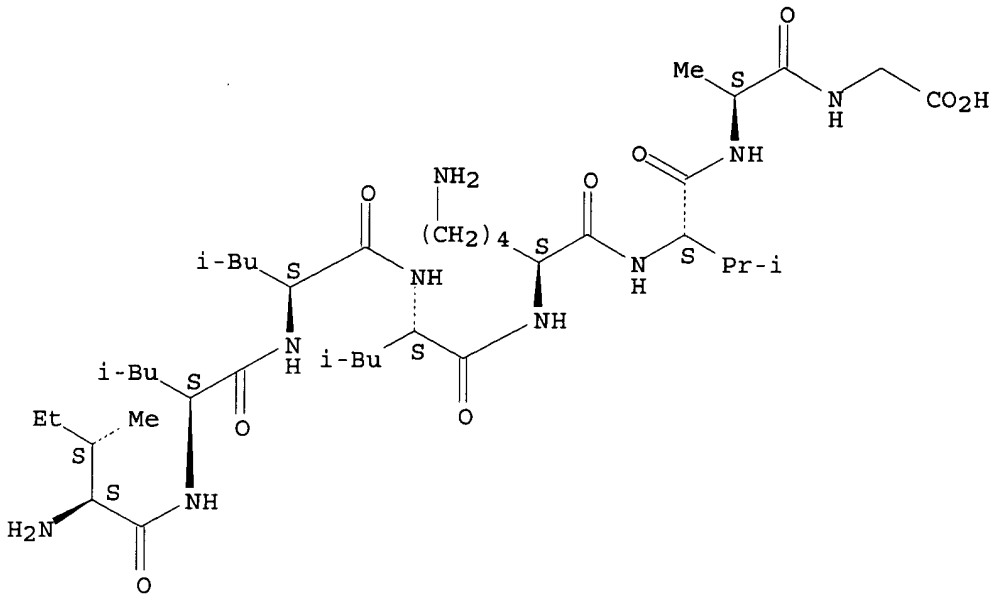
2 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 16 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **186487-61-6** REGISTRY
CN Glycine, L-isoleucyl-L-leucyl-L-leucyl-L-leucyl-L-lysyl-L-valyl-L-alanyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **8**

SEQ3 1 Ile-Leu-Leu-Leu-Lys-Val-Ala-Gly
=== ===
HITS AT: 1, 8

MF C40 H75 N9 O9
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.



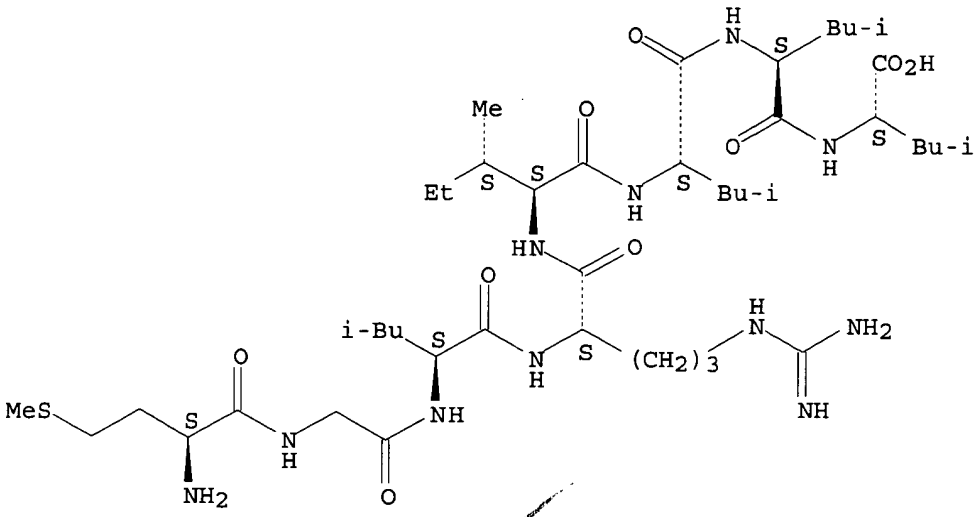
2 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 17 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **180994-66-5** REGISTRY
CN L-Leucine, L-methionylglycyl-L-leucyl-L-arginyl-L-isoleucyl-L-leucyl-L-leucyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Leucine, N-[N-[N-[N-[N2-[N-(N-L-methionylglycyl)-L-leucyl]-L-arginyl]-L-isoleucyl]-L-leucyl]-L-leucyl]-
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **8**

SEQ3 1 Met-Gly-Leu-Arg-Ile-Leu-Leu-Leu
=== ===
HITS AT: 1, 8

MF C43 H81 N11 O9 S
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



3 REFERENCES IN FILE CA (1967 TO DATE)
3 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 18 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **175800-89-2** REGISTRY
CN L-Proline, L-alanylglycyl-L-phenylalanyl-L-lysylglycyl-L-.alpha.-glutamyl-L-glutaminylglycyl-L-prolyl-L-lysylglycyl-L-.alpha.-glutamyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: WO0005249 SEQID: 2 claimed protein
CN 3: PN: WO0005250 SEQID: 3 claimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **13**

SEQ3 1 Ala-Gly-Phe-Lys-Gly-Glu-Gln-Gly-Pro-Lys-
===
11 Gly-Glu-Pro
===
HITS AT: 1, 13

MF C57 H88 N16 O19
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.

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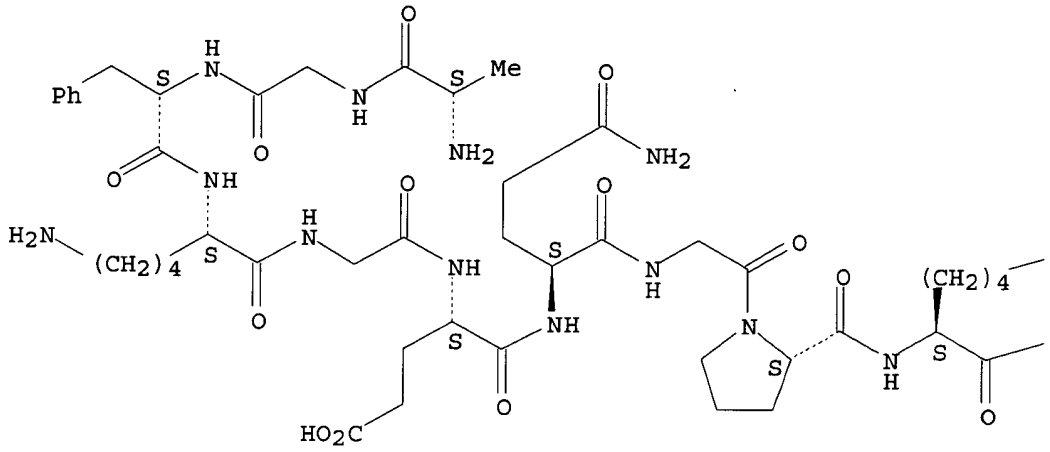

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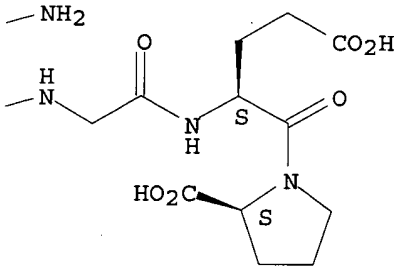


8 REFERENCES IN FILE CA (1967 TO DATE)
8 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 19 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **168650-46-2** REGISTRY
CN L-Valine, L-tyrosyl-L-methionyl-L-.alpha.-aspartylglycyl-L-threonyl-L-methionyl-L-seryl-L-glutaminyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Valine, N-[N2-[N-[N-[N-[N-[N-(N-L-tyrosyl-L-methionyl)-L-.alpha.-asparty1]glycyl]-L-threonyl]-L-methionyl]-L-seryl]-L-glutaminyl]-
OTHER NAMES:
CN 12: PN: WO0123577 SEQID: 10 claimed sequence
CN 150: PN: WO0178655 SEQID: 3 claimed sequence
CN 1: PN: WO0049041 SEQID: 20 claimed sequence

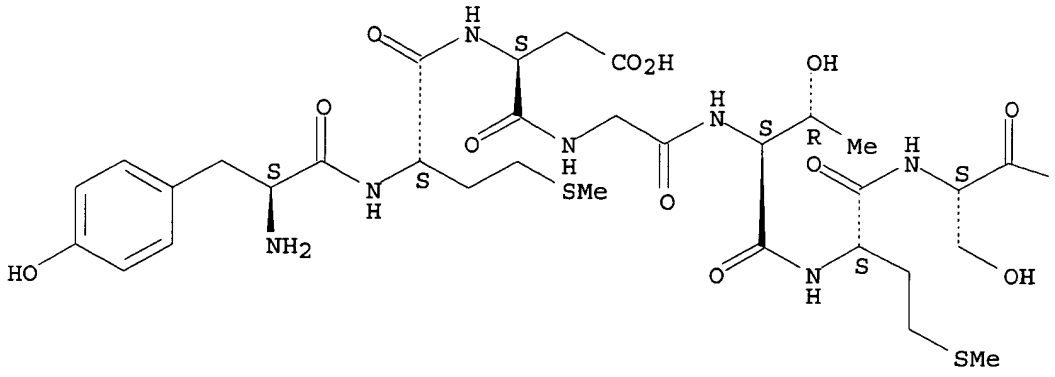
CN 22: PN: US6277956 SEQID: 22 unclaimed sequence
CN 22: PN: WO0116320 SEQID: 22 unclaimed sequence
CN 24: PN: US6326200 SEQID: 22 unclaimed sequence
CN 28: PN: WO0020445 SEQID: 18 unclaimed sequence
CN 318: PN: US20020007173 SEQID: 359 unclaimed sequence
CN 41: PN: WO0050589 SEQID: 41 unclaimed sequence
CN 42: PN: US6291430 SEQID: 78 unclaimed sequence
CN 42: PN: WO0006598 SEQID: 43 unclaimed sequence
CN 43: PN: US6245525 SEQID: 37 unclaimed sequence
CN 44: PN: WO0129220 SEQID: 42 unclaimed sequence
CN 46: PN: US6077519 SEQID: 46 claimed sequence
CN 4: PN: WO0136453 SEQID: 4 unclaimed sequence
CN 53: PN: WO0013699 SEQID: 49 unclaimed sequence
CN 58: PN: WO0078806 SEQID: 58 unclaimed sequence
CN 5: PN: WO9958678 PAGE: 62 unclaimed sequence
CN 63: PN: WO0020581 SEQID: 78 unclaimed sequence
CN 64: PN: WO0153833 SEQID: 49 unclaimed sequence
CN 65: PN: WO0006723 SEQID: 65 claimed sequence
CN 6: PN: WO0216560 SEQID: 38 unclaimed sequence
CN 75: PN: WO0032785 SEQID: 41 unclaimed sequence
CN 9: PN: WO0127291 SEQID: 10 unclaimed sequence
CN PN: WO9953061 SEQID: 37 unclaimed sequence
CN PN: WO9955892 FIGURE: 15 unclaimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Tyr-Met-Asp-Gly-Thr-Met-Ser-Gln-Val
=== ===
HITS AT: 1, 9

MF C42 H66 N10 O16 S2
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER, USPATFULL

Absolute stereochemistry.

PAGE 1-A



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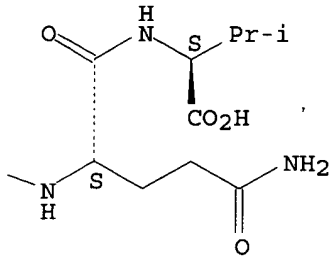


62 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
62 REFERENCES IN FILE CAPLUS (1967 TO DATE)

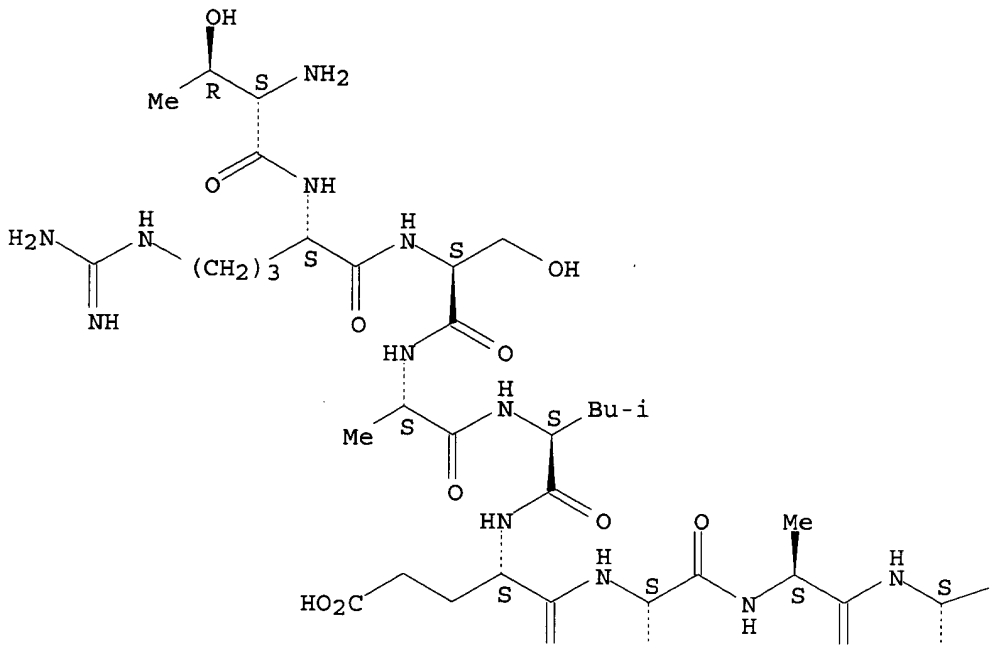
L37 ANSWER 20 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155071-14-0** REGISTRY
CN L-Alanine, N-[N-[N-[N-[N-[N-[N-(N2-L-threonyl-L-arginyl)-L-seryl]-L-alanyl]-L-leucyl]-L-.alpha.-glutamyl]-L-seryl]-L-alanyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Thr-Arg-Ser-Ala-Leu-Glu-Ser-Ala-Ala
=== ===
HITS AT: 1, 9

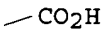
MF C36 H64 N12 O15
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



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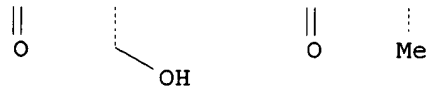


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

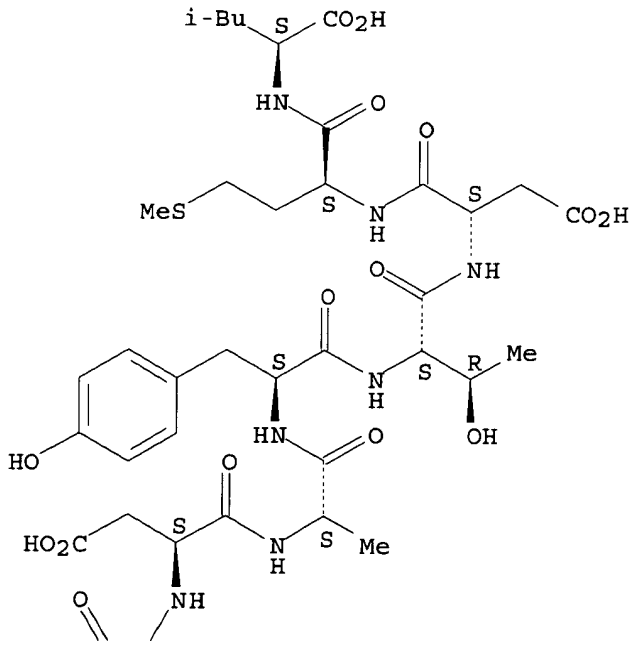
L37 ANSWER 21 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155071-13-9** REGISTRY
CN L-Leucine, N-[N-[N-[N-[N-[N-(N2-L-leucyl-L-arginyl)-L-.alpha.-aspartyl]-L-alanyl]-L-tyrosyl]-L-threonyl]-L-.alpha.-aspartyl]-L-methionyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Leu-Arg-Asp-Ala-Tyr-Thr-Asp-Met-Leu
=== ===
HITS AT: 1, 9

MF C47 H76 N12 O16 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



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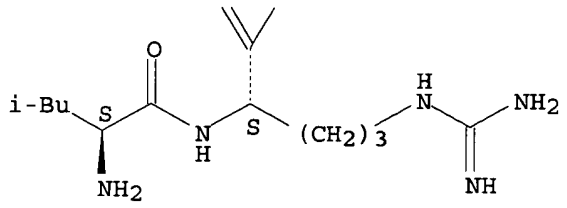

2 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 22 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155071-11-7** REGISTRY
CN L-Serine, N-[N-[N-[N2-[N-[N-[N-(N2-L-alanyl-L-arginyl)-L-isoleucyl]-L-valyl]-L-leucyl]-L-lysyl]-L-alanyl]-L-leucyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ala-Arg-Ile-Val-Leu-Lys-Ala-Leu-Ser
=== ===
HITS AT: 1, 9

MF C44 H83 N13 O11
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

Mol. Immunol. 31 (5) 331-6 1994

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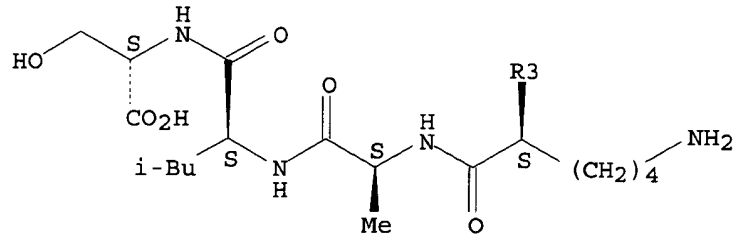

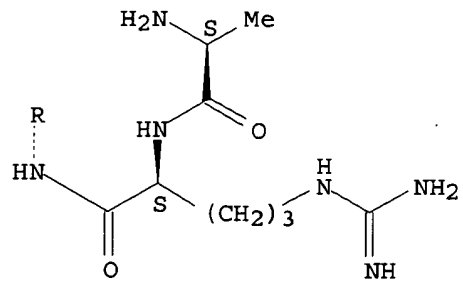

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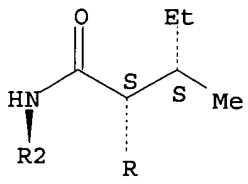

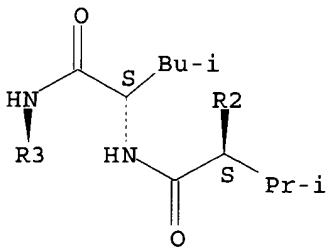

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 23 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155071-10-6** REGISTRY
CN L-Alanine, N-[N-[N-[N-[1-[N-[N-(N2-L-isoleucyl-L-arginyl)-L-cysteinyl]-L-isoleucyl]-L-prolyl]-L-threonyl]-L-leucyl]-L-.alpha.-glutamyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ile-Arg-Cys-Ile-Pro-Thr-Leu-Glu-Ala
=== ===
HITS AT: 1, 9

MF C44 H78 N12 O13 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

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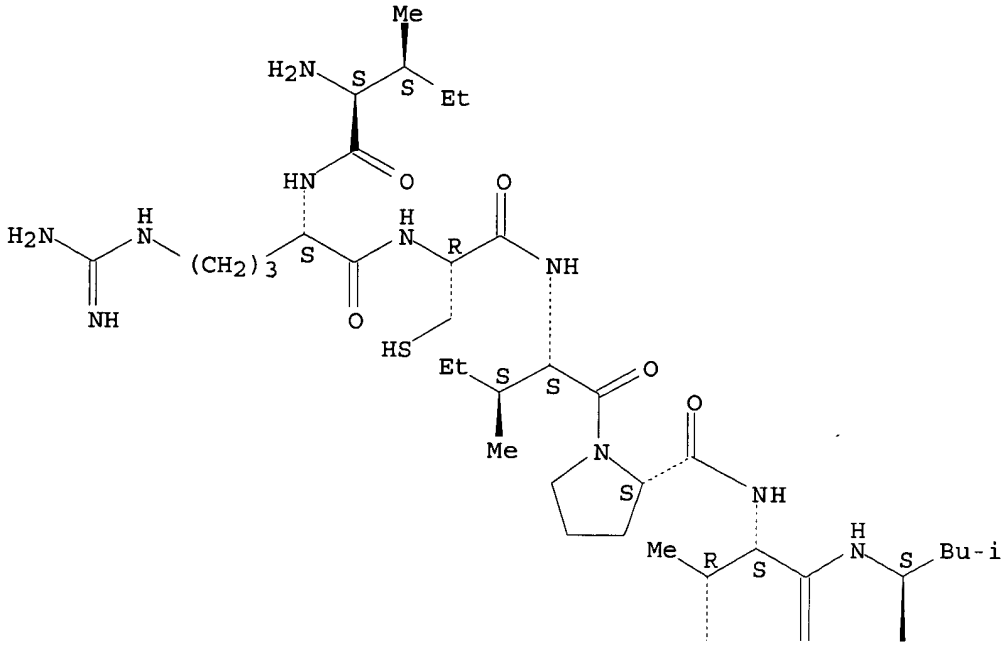

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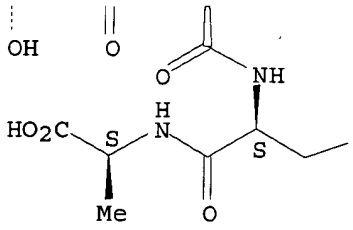

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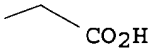


1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 24 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155071-08-2** REGISTRY
CN L-Isoleucine, N-[N-[N-[N-[N-[N-[N-(N2-L-isoleucyl-L-arginyl)-L-valyl]glycyl]-L-alanyl]-L-alanyl]-L-threonyl]-L-.alpha.-glutamyl]- (9CI) (CA INDEX NAME)

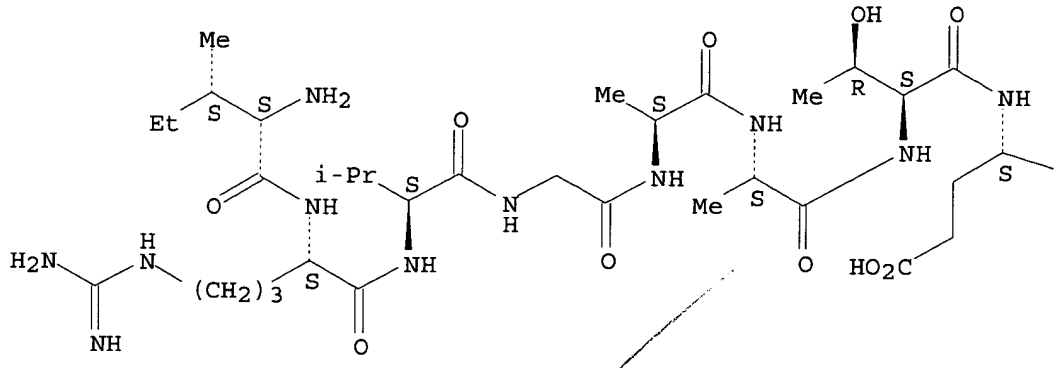
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ile-Arg-Val-Gly-Ala-Ala-Thr-Glu-Ile
=== ===
HITS AT: 1, 9

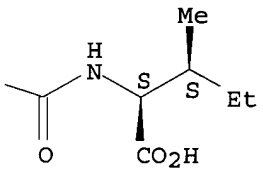
MF C40 H72 N12 O13
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 25 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155071-02-6** REGISTRY
CN L-Alanine, N-[N-[N-[N2-[N-[N-[N-(N2-L-isoleucyl-L-arginyl)glycyl]glycyl]-L-phenylalanyl]-L-arginyl]-L-valyl]-L-cysteinyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ile-Arg-Gly-Gly-Phe-Arg-Val-Cys-Ala
=== ===
HITS AT: 1, 9

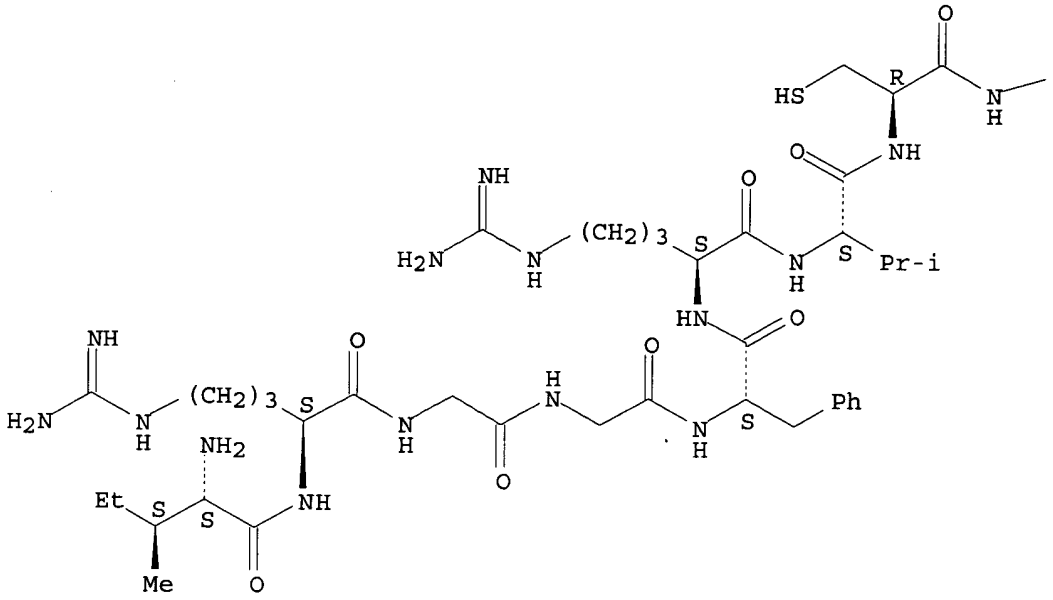
MF C42 H71 N15 O10 S
SR CA

Mol. Immunol. 31 (5) 331-336 1994

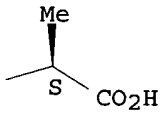
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

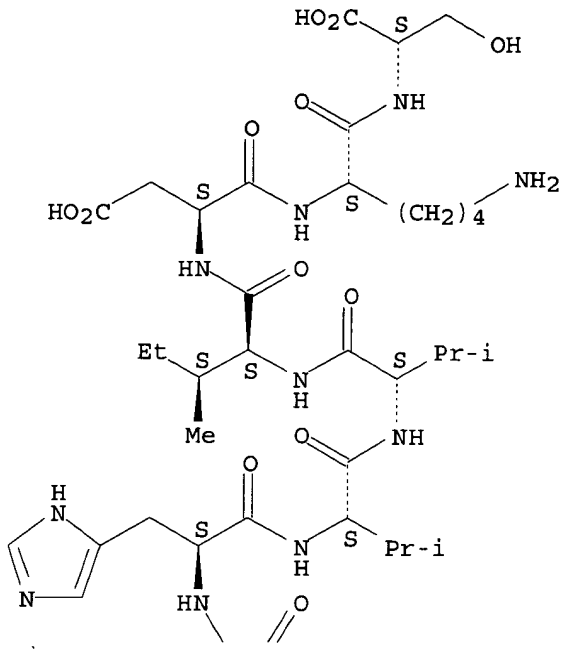
L37 ANSWER 26 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155070-96-5** REGISTRY
CN L-Serine, N-[N2-[N-[N-[N-[N-[N-(N2-glycyl-L-arginyl)-L-histidyl]-L-valyl]-L-valyl]-L-isoleucyl]-L-.alpha.-aspartyl]-L-lysyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Gly-Arg-His-Val-Val-Ile-Asp-Lys-Ser
=== ===
HITS AT: 1, 9

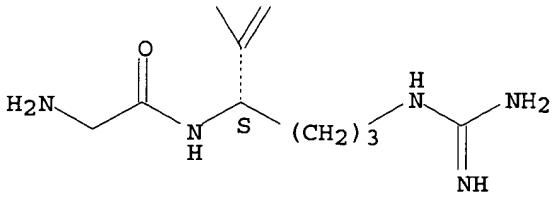
MF C43 H75 N15 O13
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 2-A



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

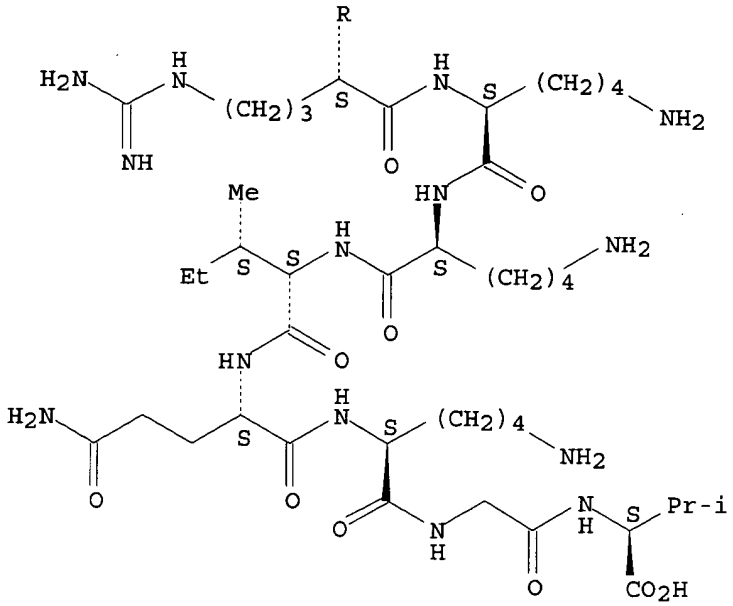
L37 ANSWER 27 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **155070-95-4** REGISTRY
CN L-Valine, N-[N-[N2-[N2-[N-[N-[N2-[N2-(N2-L-alanyl-L-arginyl)-L-lysyl]-L-lysyl]-L-isoleucyl]-L-glutaminyl]-L-lysyl]glycyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ala-Arg-Lys-Lys-Ile-Gln-Lys-Gly-Val
=== ===
HITS AT: 1, 9

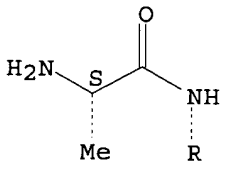
MF C45 H86 N16 O11
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 2-A



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

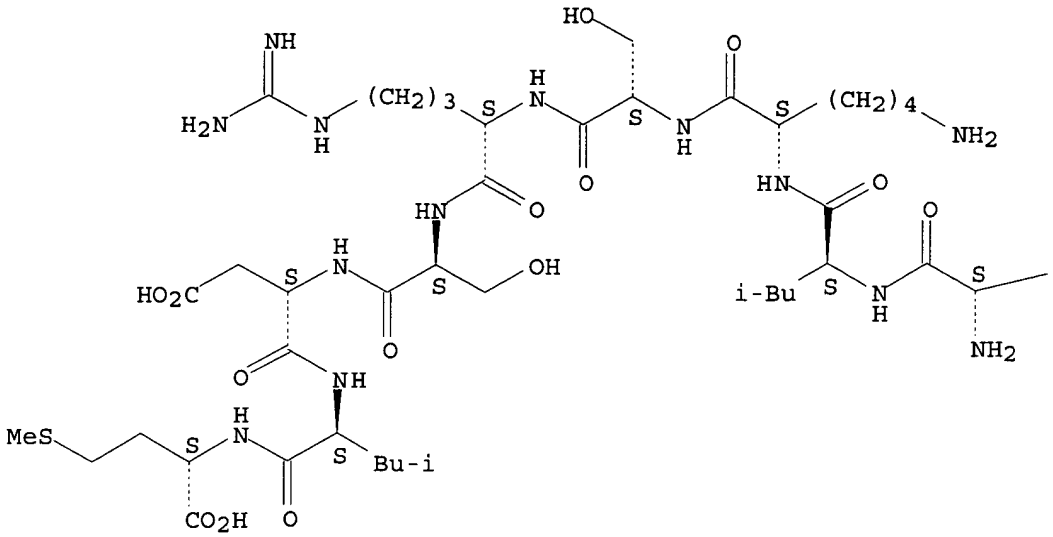
L37 ANSWER 28 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **152244-26-3** REGISTRY
CN L-Methionine, N-[N-[N-[N-[N2-[N-[N2-(N-L-isoleucyl-L-leucyl)-L-lysyl]-L-seryl]-L-arginyl]-L-seryl]-L-.alpha.-aspartyl]-L-leucyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Ile-Leu-Lys-Ser-Arg-Ser-Asp-Leu-Met
=== ===
HITS AT: 1, 9

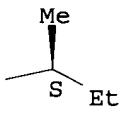
MF C45 H83 N13 O14 S
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

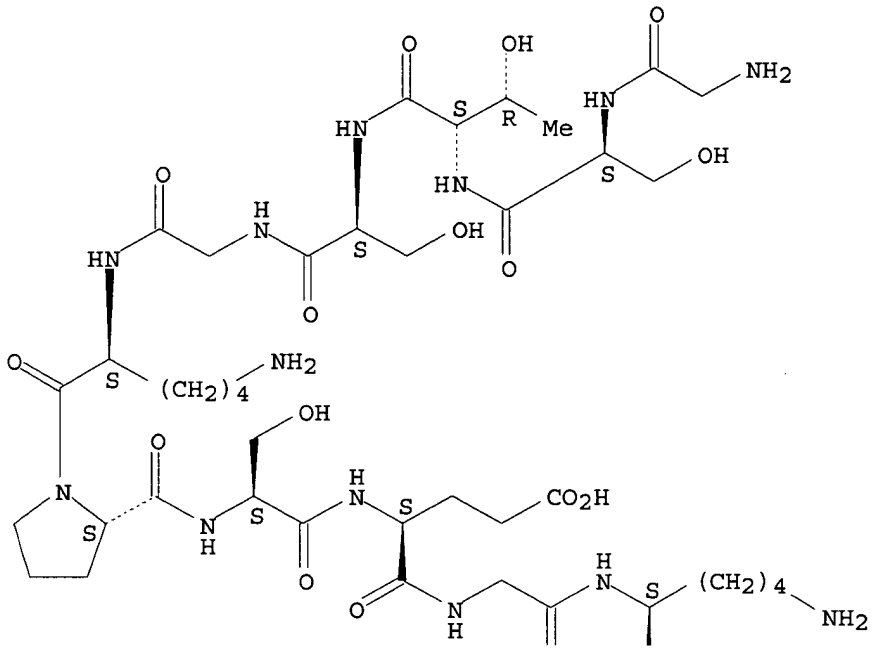
L37 ANSWER 29 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **150243-60-0** REGISTRY
CN Glycine, glycyl-L-seryl-L-threonyl-L-serylglycyl-L-lysyl-L-prolyl-L-seryl-L-.alpha.-glutamylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Glycine, N-[N2-[N-[N-[N-[1-[N2-[N-[N-[N-(N-glycyl-L-seryl)-L-threonyl]-L-seryl]glycyl]-L-lysyl]-L-prolyl]-L-seryl]-L-.alpha.-glutamyl]glycyl]-L-lysyl]-
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **12**

SEQ3 1 Gly-Ser-Thr-Ser-Gly-Lys-Pro-Ser-Glu-Gly-
=== 11 Lys-Gly
===
HITS AT: 1, 12

MF C43 H74 N14 O19
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.

PAGE 1-A



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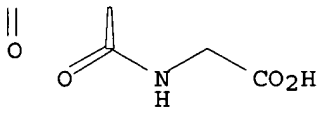


2 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 30 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **150243-58-6** REGISTRY
CN L-Serine, glycyl-L-lysyl-L-seryl-L-serylglycyl-L-serylglycyl-L-seryl-L-.alpha.-glutamyl-L-seryl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Serine, N-[N2-[N-[N-[N-[N-[N-[N-[N-(N2-glycyl-L-lysyl)-L-seryl]-L-seryl]glycyl]-L-seryl]glycyl]-L-seryl]-L-.alpha.-glutamyl]-L-seryl]-L-lysyl]-
OTHER NAMES:
CN 2: PN: WO0147512 SEQID: 3 claimed sequence
CN 2: PN: WO0210405 SEQID: 13 unclaimed sequence
CN 36: PN: WO0210406 SEQID: 33 unclaimed sequence
CN 3: PN: WO0004926 SEQID: 3 unclaimed protein

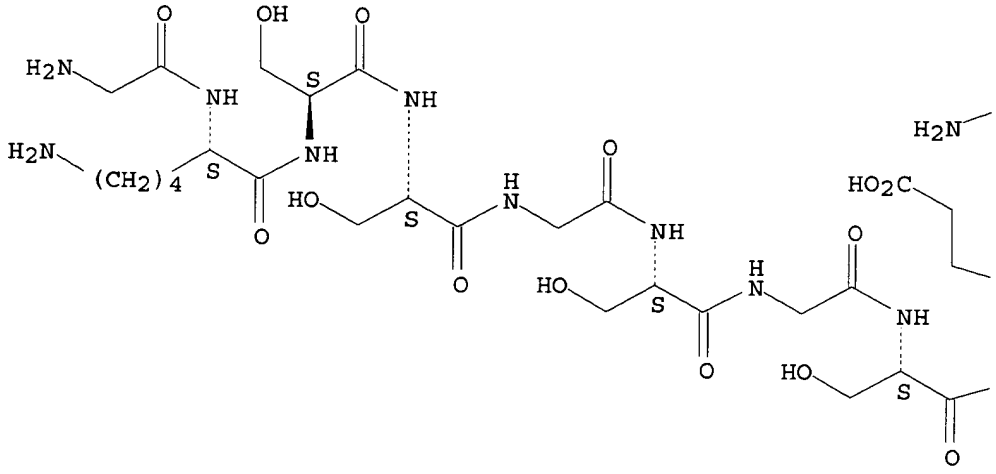
CN 3: PN: WO0021576 SEQID: 3 claimed protein
CN 3: PN: WO0136447 PAGE: 13 unclaimed sequence
CN 59: PN: WO0071565 SEQID: 57 unclaimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **12**

SEQ3 1 Gly-Lys-Ser-Ser-Gly-Ser-Gly-Ser-Glu-Ser-
===
11 Lys-Ser
===
HITS AT: 1, 12

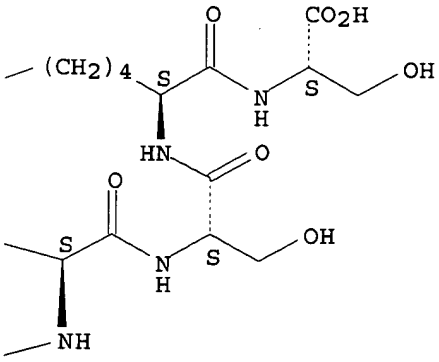
MF C41 H72 N14 O21
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B



11 REFERENCES IN FILE CA (1967 TO DATE)

11 REFERENCES IN FILE CAPLUS (1967 TO DATE)

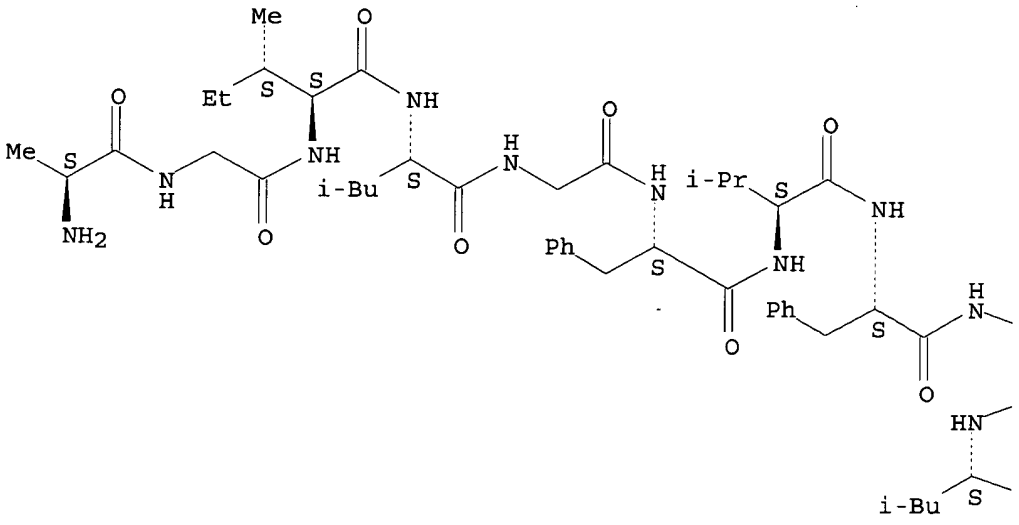
L37 ANSWER 31 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **148138-87-8** REGISTRY
CN L-Valine, N-[N-[N-[N-[N-[N-[N-[N-[N-(N-L-alanylglycyl)-L-isoleucyl]-L-leucyl]glycyl]-L-phenylalanyl]-L-valyl]-L-phenylalanyl]-L-threonyl]-L-leucyl]-L-threonyl]- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **12**

SEQ3 1 Ala-Gly-Ile-Leu-Gly-Phe-Val-Phe-Thr-Leu-
===
11 Thr-Val
===
HITS AT: 1, 12

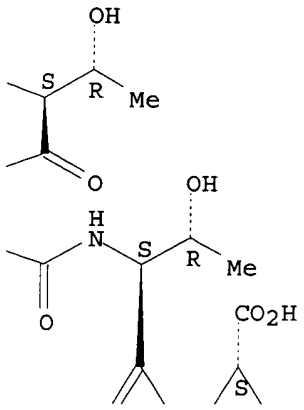
MF C61 H96 N12 O15
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER

Absolute stereochemistry.

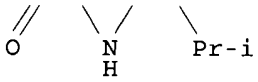
PAGE 1-A



PAGE 1-B



PAGE 2-B



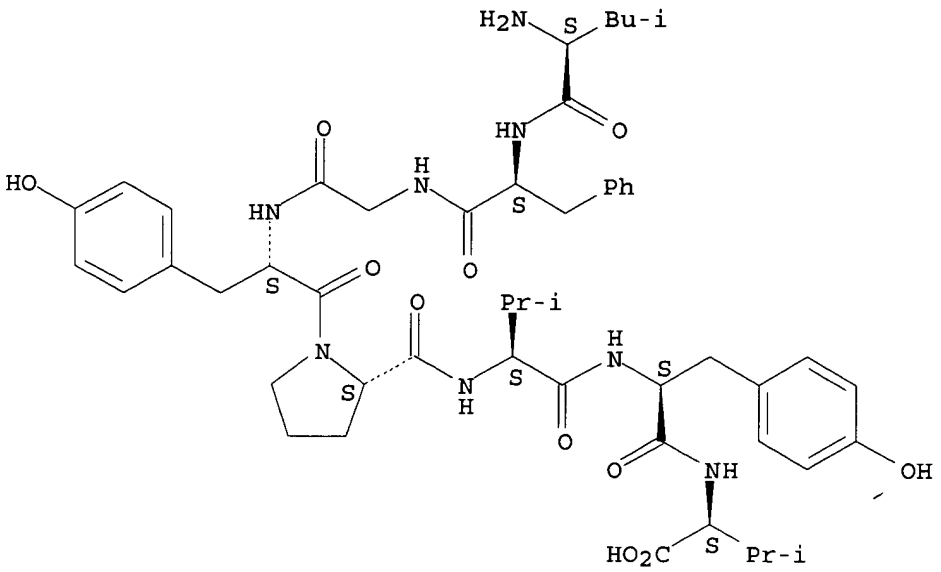
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 32 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **141997-16-2** REGISTRY
CN L-Valine, L-leucyl-L-phenylalanylglycyl-L-tyrosyl-L-prolyl-L-valyl-L-tyrosyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Valine, N-[N-[N-[1-[N-[N-(N-L-leucyl-L-phenylalanyl)glycyl]-L-tyrosyl]-L-prolyl]-L-valyl]-L-tyrosyl]-
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **8**

SEQ3 1 Leu-Phe-Gly-Tyr-Pro-Val-Tyr-Val
=== ===
HITS AT: 1, 8

MF C50 H68 N8 O11
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER

Absolute stereochemistry.



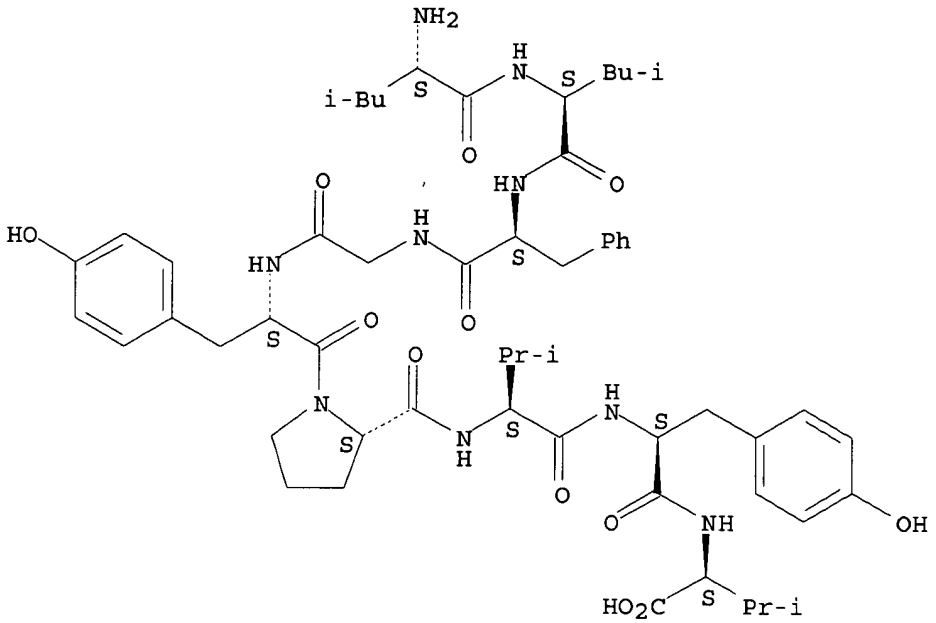
6 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
6 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 33 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **141677-18-1** REGISTRY
CN L-Valine, L-leucyl-L-leucyl-L-phenylalanylglycyl-L-tyrosyl-L-prolyl-L-valyl-L-tyrosyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Valine, N-[N-[N-[1-[N-[N-[N-(N-L-leucyl-L-leucyl)-L-phenylalanyl]glycyl]-L-tyrosyl]-L-prolyl]-L-valyl]-L-tyrosyl]-
OTHER NAMES:
CN 10: PN: WO9960119 PAGE: 79 unclaimed sequence
CN 152: PN: WO0100225 TABLE: 6 claimed sequence
CN 17: PN: WO9964597 SEQID: 17 unclaimed sequence
CN 18: PN: WO0025813 SEQID: 18 unclaimed sequence
CN 244: PN: US20020007173 SEQID: 285 unclaimed sequence
CN 3: PN: WO0172768 SEQID: 3 unclaimed sequence
CN 5: PN: WO0071158 PAGE: 4 claimed protein
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

SEQ3 1 Leu-Leu-Phe-Gly-Tyr-Pro-Val-Tyr-Val
=== ===
HITS AT: 1, 9

MF C56 H79 N9 O12
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



46 REFERENCES IN FILE CA (1967 TO DATE)
5 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
46 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 34 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **141368-69-6** REGISTRY
CN L-Leucine, glycyl-L-isoleucyl-L-leucylglycyl-L-phenylalanyl-L-valyl-L-phenylalanyl-L-threonyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Leucine, N-[N-[N-[N-[N-[N-(N-glycyl-L-isoleucyl)-L-leucyl]glycyl]-L-phenylalanyl]-L-valyl]-L-phenylalanyl]-L-threonyl]-
OTHER NAMES:
CN 1: PN: WO0194944 SEQID: 43 claimed protein
CN 1: PN: WO0216560 SEQID: 33 unclaimed sequence
CN 20: PN: DE19925199 SEQID: 13 unclaimed sequence
CN 20: PN: WO0025813 SEQID: 20 unclaimed sequence
CN 20: PN: WO0127291 SEQID: 21 unclaimed sequence
CN 23: PN: WO0123577 SEQID: 21 claimed sequence
CN 245: PN: US20020007173 SEQID: 286 unclaimed sequence
CN 26: PN: US6183746 SEQID: 24 unclaimed sequence
CN 4: PN: US5989565 SEQID: 4 unclaimed sequence
CN 4: PN: US6077519 SEQID: 4 unclaimed sequence
CN 53: PN: WO0049041 SEQID: 57 claimed sequence
CN 5: PN: WO0023053 SEQID: 5 claimed protein
CN 5: PN: WO0136453 SEQID: 5 unclaimed sequence
CN 61: PN: WO0024778 SEQID: 57 unclaimed sequence
CN 6: PN: WO0180833 SEQID: 12 unclaimed sequence
CN 75: PN: WO0118035 PAGE: 36 unclaimed sequence
CN 8: PN: WO0124829 PAGE: 24 claimed protein
CN 9: PN: WO9960119 PAGE: 79 unclaimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

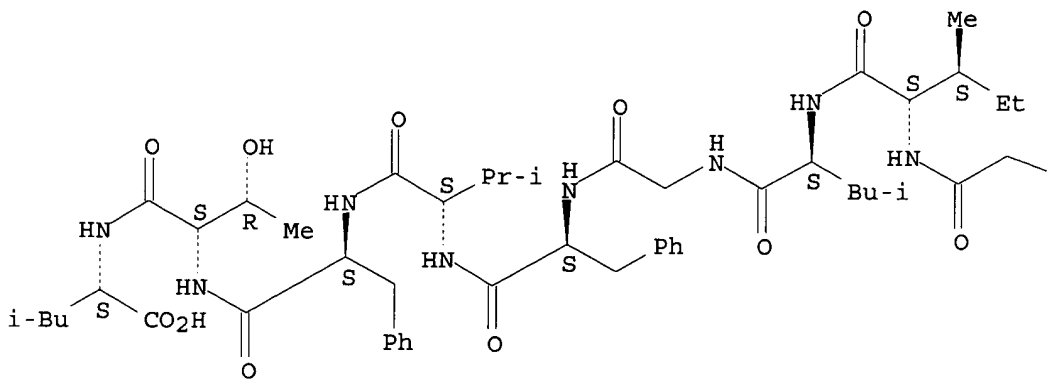
SEQ3 1 Gly-Ile-Leu-Gly-Phe-Val-Phe-Thr-Leu
=== ===

HITS AT: 1, 9

MF C49 H75 N9 O11
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER, USPATFULL

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B

104 REFERENCES IN FILE CA (1967 TO DATE)
6 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
104 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 35 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **139079-41-7** REGISTRY
CN L-Valine, L-isoleucyl-L-leucyl-L-lysyl-L-.alpha.-glutamyl-L-prolyl-L-valyl-L-histidylglycyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Valine, N-[N-[N-[N-[1-[N-[N2-(N-L-isoleucyl-L-leucyl)-L-lysyl]-L-.alpha.-glutamyl]-L-prolyl]-L-valyl]-L-histidyl]glycyl]-
OTHER NAMES:
CN 19: PN: TABLE: 1 claimed protein
CN 1: PN: EP1088889 SEQID: 1 claimed protein
CN 20: PN: WO0147955 SEQID: 21 claimed protein
CN 21: PN: WO0226254 SEQID: 21 claimed protein
CN 22: PN: WO0127291 SEQID: 23 unclaimed sequence
CN 256: PN: WO9958658 TABLE: 6 claimed protein
CN 25: PN: WO0123577 SEQID: 23 claimed sequence
CN 319: PN: US20020007173 SEQID: 360 unclaimed sequence
CN 391: PN: WO9958658 SEQID: 101 unclaimed protein
CN 7: PN: WO0124829 PAGE: 20 claimed protein
CN 7: PN: WO0136452 FIGURE: 1a unclaimed sequence

CN PN: US5976551 SEQID: 145 claimed sequence
CN PN: US5981706 SEQID: 62 claimed protein
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **9**

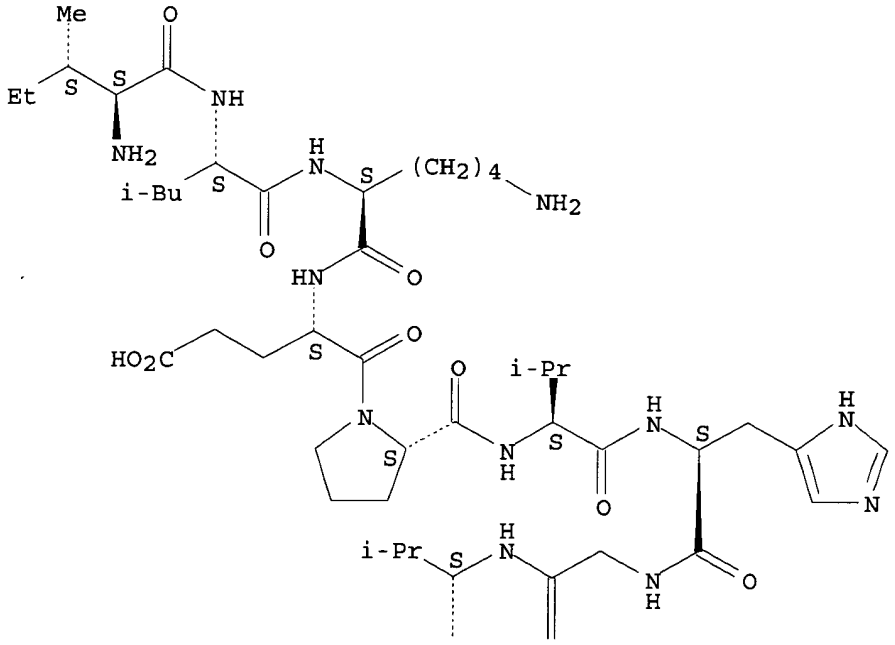
SEQ3 1 Ile-Leu-Lys-Glu-Pro-Val-His-Gly-Val
=== ===
HITS AT: 1, 9

MF C46 H78 N12 O12
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

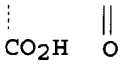
WO 9420127 J. Exp. Med (1994) 180 (4) 1283-93

Absolute stereochemistry.

PAGE 1-A



PAGE 2-A



96 REFERENCES IN FILE CA (1967 TO DATE)
11 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
96 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 36 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **138831-86-4** REGISTRY
CN L-Leucine, L-seryl-L-isoleucyl-L-isoleucyl-L-asparaginyl-L-phenylalanyl-L-.alpha.-glutamyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Leucine, N-[N2-[N-[N-[N2-[N-(N-L-seryl-L-isoleucyl)-L-isoleucyl]-L-

asparaginyl]-L-phenylalanyl]-L-.alpha.-glutamyl]-L-lysyl]-
OTHER NAMES:
CN 10: PN: US20020018785 SEQID: 10 unclaimed sequence
CN 11: PN: WO0135991 SEQID: 11 claimed protein
CN 11: PN: WO0157068 SEQID: 11 unclaimed sequence
CN 11: PN: WO0172995 SEQID: 26 unclaimed sequence
CN 1: PN: US6210672 SEQID: 1 claimed sequence
CN 1: PN: WO0034494 PAGE: 80 unclaimed sequence
CN 246: PN: WO0069900 SEQID: 1550 unclaimed sequence
CN 2: PN: WO0140275 SEQID: 2 claimed protein
CN 2: PN: WO0179510 PAGE: 25 unclaimed sequence
CN 35: PN: WO0178768 SEQID: 35 unclaimed sequence
CN 3: PN: WO0172123 SEQID: 4 unclaimed sequence
CN 48: PN: WO9958658 SEQID: 97 unclaimed protein
CN 4: PN: WO0071158 PAGE: 4 claimed protein
CN 4: PN: WO0154720 PAGE: 15 claimed protein
CN 4: PN: WO0179259 SEQID: 5 unclaimed sequence
CN 6: PN: WO0160404 SEQID: 6 unclaimed sequence
CN 7: PN: WO0174855 SEQID: 22 unclaimed sequence
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL 8

SEQ3 1 Ser-Ile-Ile-Asn-Phe-Glu-Lys-Leu
=== ===
HITS AT: 1, 8

MF C45 H74 N10 O13
SR CA
LC STN Files: CA, CAPLUS, CHEMCATS, TOXCENTER, USPATFULL

Absolute stereochemistry.

J. Biol. Chem. 1993 268(21) 15425-34

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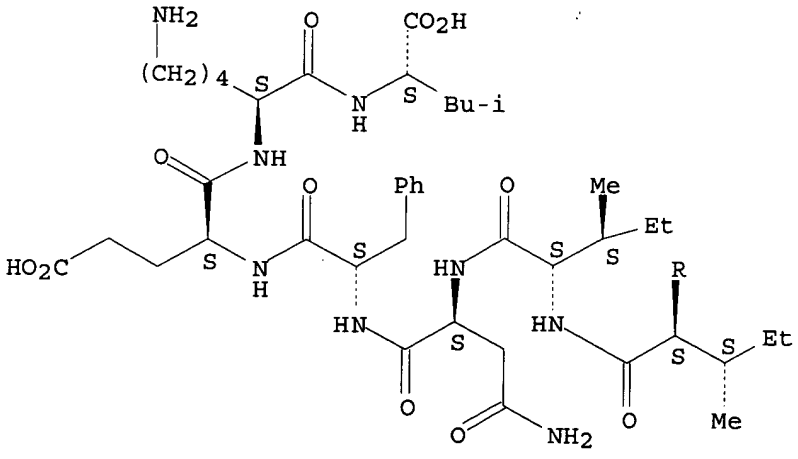
Cell 1993 73(5) 1041-9



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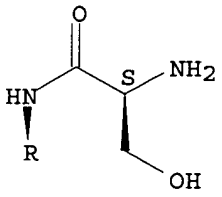

136 REFERENCES IN FILE CA (1967 TO DATE)
16 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
136 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 37 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **124470-30-0** REGISTRY
CN L-Alanine, L-prolyl-L-seryl-L-glutaminyl-L-arginyl-L-histidylglycyl-L-seryl-L-lysyl-L-tyrosyl-L-leucyl-L-alanyl-L-threonyl- (9CI) (CA INDEX NAME)
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **13**

SEQ3 1 Pro-Ser-Gln-Arg-His-Gly-Ser-Lys-Tyr-Leu-
=== 11 Ala-Thr-Ala
===

HITS AT: 1, 13

MF C61 H98 N20 O19
SR CA
LC STN Files: CA, CAPLUS

Absolute stereochemistry.

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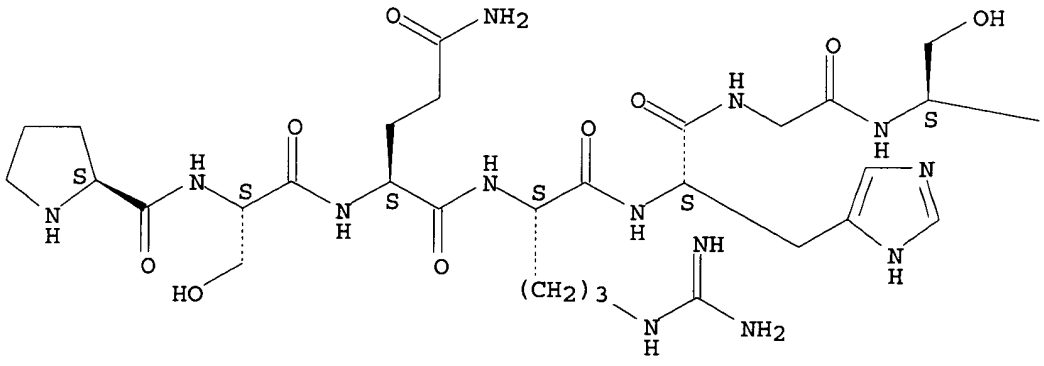

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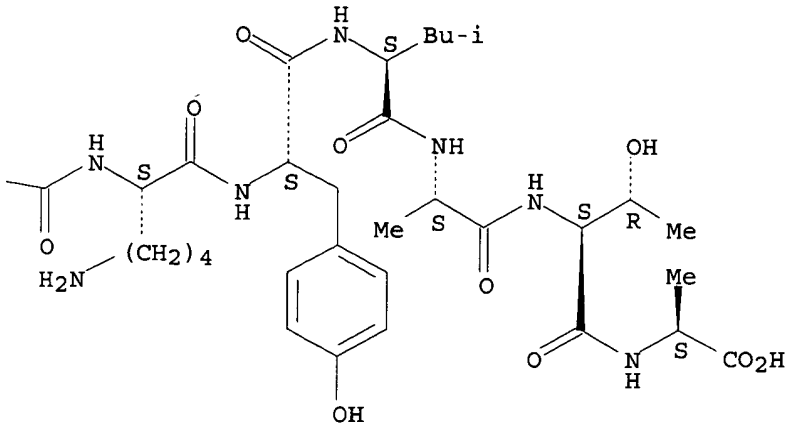

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 38 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **122630-93-7** REGISTRY
CN L-Threonine, L-prolyl-L-lysyl-L-tyrosyl-L-valyl-L-lysyl-L-glutaminyl-L-asparaginyl-L-threonyl-L-leucyl-L-lysyl-L-leucyl-L-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 10: PN: WO0121189 TABLE: 5 unclaimed sequence
CN 11: PN: WO0141787 TABLE: 5 unclaimed sequence
CN 126: PN: WO0141788 TABLE: 5 unclaimed sequence
CN 13: PN: WO0142267 TABLE: 5 unclaimed sequence
CN 14: PN: WO0145728 TABLE: 5 unclaimed sequence
CN 152: PN: WO0142270 TABLE: 5 unclaimed sequence
CN 1: PN: US6230102 SEQID: 1 unclaimed sequence
CN 1: PN: WO0005249 SEQID: 1 claimed protein
CN 1: PN: WO0044887 SEQID: 1 unclaimed sequence
CN 1: PN: WO0072876 PAGE: 43 unclaimed sequence
CN 1: PN: WO0072880 PAGE: 28 unclaimed sequence
CN 20: PN: WO0219986 TABLE: 5 claimed sequence
CN 2: PN: WO0005250 SEQID: 2 claimed sequence
CN 409: PN: WO0032785 SEQID: 147 claimed sequence
CN 4: PN: WO0149821 PAGE: 13 unclaimed sequence
CN 6: PN: WO0222860 SEQID: 3 claimed sequence
CN 78: PN: WO0141799 PAGE: 99 unclaimed sequence
CN 8: PN: WO0023053 SEQID: 8 claimed protein
CN 8: PN: WO0180833 SEQID: 14 unclaimed sequence
CN HA 307-319
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **13**

SEQ3 1 Pro-Lys-Tyr-Val-Lys-Gln-Asn-Thr-Leu-Lys-
=== 11 Leu-Ala-Thr
===
HITS AT: 1, 13

MF C69 H118 N18 O19
SR CA

LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.

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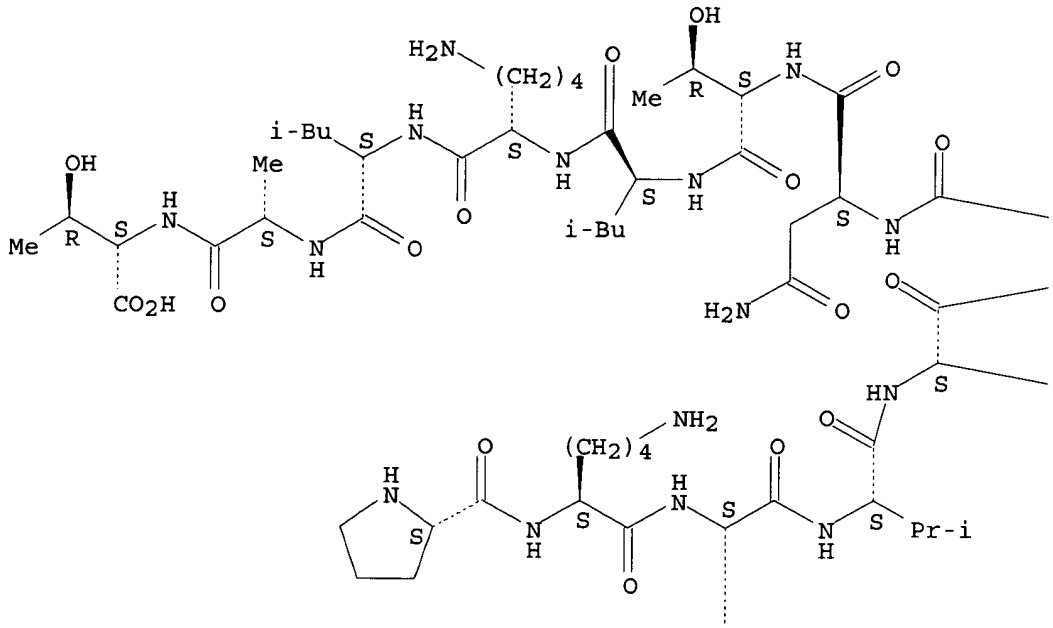

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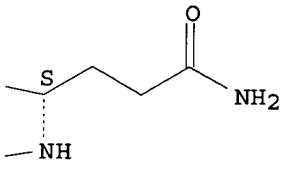

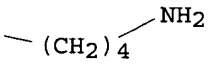

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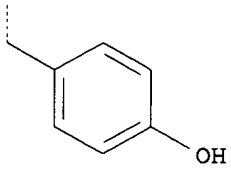

96 REFERENCES IN FILE CA (1967 TO DATE)
10 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
96 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L37 ANSWER 39 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **84745-13-1** REGISTRY
CN L-Serine, L-prolyl-L-leucyl-L-seryl-L-arginyl-L-threonyl-L-leucyl-L-seryl-L-valyl-L-seryl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN L-Serine, N-[N-[N-[N-[N-[N-[N2-[N-(N-L-prolyl-L-leucyl)-L-seryl]-L-arginyl]-L-threonyl]-L-leucyl]-L-seryl]-L-valyl]-L-seryl]-
OTHER NAMES:
CN 28: PN: WO0224947 SEQID: 28 unclaimed sequence
CN 36: PN: WO0069900 SEQID: 1337 unclaimed sequence
CN 3: PN: WO0107638 PAGE: 10 unclaimed sequence
CN N-syntide
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL **10**

SEQ3 1 Pro-Leu-Ser-Arg-Thr-Leu-Ser-Val-Ser-Ser
=== ===
HITS AT: 1, 10

MF C44 H79 N13 O16
LC STN Files: CA, CAPLUS, TOXCENTER

Absolute stereochemistry.

91 89613

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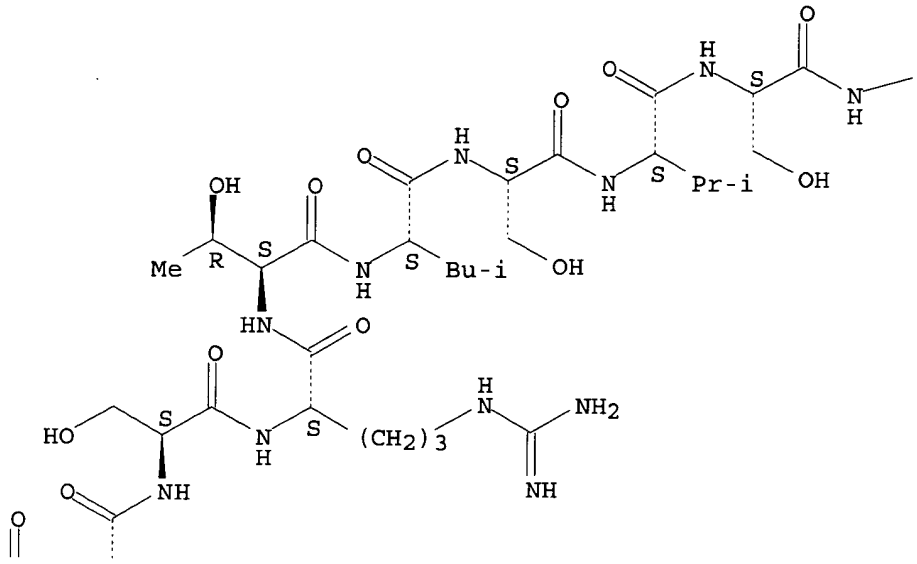

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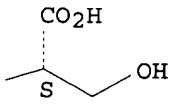

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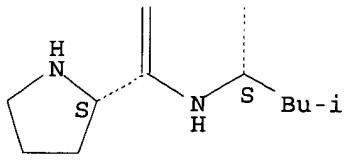


9 REFERENCES IN FILE CA (1967 TO DATE)
9 REFERENCES IN FILE CAPLUS (1967 TO DATE)

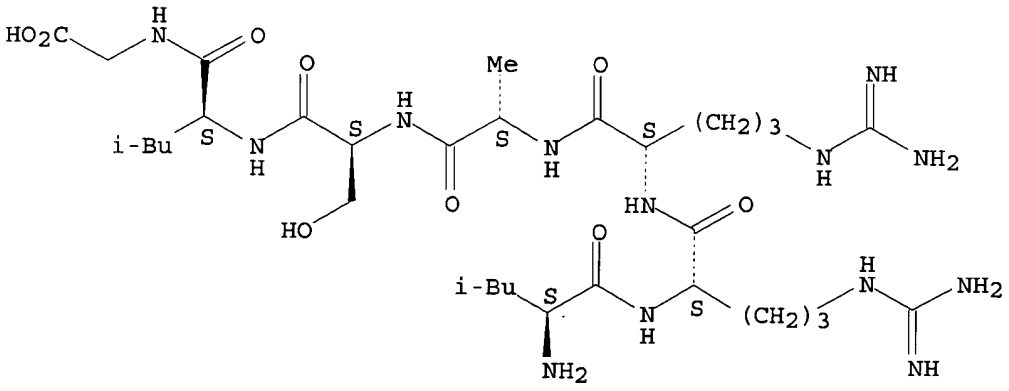
L37 ANSWER 40 OF 40 REGISTRY COPYRIGHT 2002 ACS
RN **65189-71-1** REGISTRY
CN Glycine, L-leucyl-L-arginyl-L-arginyl-L-alanyl-L-seryl-L-leucyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Glycine, N-[N-[N-[N-[N2-(N2-L-leucyl-L-arginyl)-L-arginyl]-L-alanyl]-L-

seryl]-L-leucyl]-
OTHER NAMES:
CN 12: PN: US6255069 SEQID: 12 claimed sequence
CN 14: PN: US6258776 SEQID: 23 unclaimed sequence
CN 16: PN: WO0146694 SEQID: 16 unclaimed sequence
CN 1: PN: US6335176 SEQID: 2 unclaimed sequence
CN 1: PN: WO0207721 PAGE: 41 unclaimed sequence
CN 2: PN: US6291214 SEQID: 14 unclaimed sequence
CN 4: PN: US6004757 SEQID: 16 unclaimed protein
CN Kemptide
FS PROTEIN SEQUENCE; STEREOSEARCH
SQL 7

SEQ3 1 Leu-Arg-Arg-Ala-Ser-Leu-Gly
=== ===
HITS AT: 1, 7

MF C32 H61 N13 O9
CI COM
LC STN Files: AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CHEMCATS, CSCHEM, EMBASE, MEDLINE, MSDS-OHS, TOXCENTER, USPATFULL

Absolute stereochemistry.



220 REFERENCES IN FILE CA (1967 TO DATE)
15 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
220 REFERENCES IN FILE CAPLUS (1967 TO DATE)